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RCRA Facility Investigation



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CIBA Site
Cranston, Rhode Island

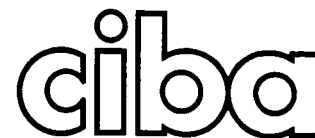
On-Site Interim Remedial Measures Work Plan (Production and Warwick Areas)

Prepared For:
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Regional Remediation
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March 1995
Project No. 87X4660

Regional Remediation Team



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March 13, 1995

Mr. Frank Battaglia, Project Manager
United States Environmental Protection Agency - Region I
90 Canal Street, Waste Management Building
Boston, Massachusetts 02114

**RE: ON-SITE IRM WORK PLAN
CIBA-GEIGY SITE, CRANSTON, RHODE ISLAND**

Dear Mr. Battaglia:

Ciba, Woodward Clyde Consultants, and PTRL Environmental Services are pleased to submit this On-Site Interim Remedial Measures (IRM) Work Plan for your review and comment. We propose to remove PCB contaminated soil from the Production Area to a target cleanup level of 45 PPM (total PCBs) and from the Warwick Property, to 1 PPM (total PCB) and dispose of the material at a TSCA regulated landfill. The risk assessment provided as Appendix A of the Work Plan clearly shows that these target cleanup levels would be fully protective of the respective land uses, based upon Media Protection Standards (MPS) for total PCBs.

With the submittal of this Work Plan, we intend to apply for the appropriate equivalent permits, select a contractor(s) and plan for premobilization activities, as show on Figure 8-2. The actual removals should start in early May and be completed by mid-June so that construction of the Stabilization Project in the Production Area can continue, as scheduled. This schedule is extremely tight such that any delay will impact the construction of the stabilization project. The work on the Warwick Property would have no effect on this schedule. Ciba intends to implement this IRM on a voluntary basis but with the clear belief that it will more than satisfy the remedial requirements for soil in both areas.

If there are any questions regarding this Work Plan, please call me at 908-914-2715.

Very truly yours,

A handwritten signature in cursive script that reads "Barry J. Berdahl".

Barry J. Berdahl Ph.D, C.H.M.M.
Regional Compliance Manager

cc: Mayor M. Traficante, City of Cranston
Mr. J. Unsworth, RIDEM

RCRA Facility Investigation

**CIBA Site
Cranston, Rhode Island**

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INTRODUCTION

1.1 OVERVIEW

This Interim Remedial Measures Work Plan describes the work that will be performed in excavating soil in the Production Area and in the Warwick Area (SWMU-5 and SWMU-6) at the former Ciba-Geigy Corporation (Ciba) facility (hereafter called the "Site") at Cranston, Rhode Island. This chapter reviews the histories of the Site, the project, the stabilization investigation, and these Interim Remedial Measures (IRMs). This chapter also describes the integration of the IRMs with stabilization and the Corrective Measures Study. Finally, the objectives of the IRMs are presented along with a discussion on the organization of this Work Plan.

1.2 BACKGROUND

This section briefly reviews the history of the Site and the history of the project. A more detailed history of the project and the Site was presented in Chapter 1 of the Phase I Interim Report (submitted in November 1991).

1.2.1 History of the Site

The Alrose Chemical Company manufactured chemicals at the Site starting in 1930. After the Geigy Chemical Company of New York purchased the Site in 1954 and merged with the Ciba Corporation in 1970, the Site was used for batch manufacturing of organic chemicals. Agricultural products, leather and textile auxiliaries, plastic additives, optical brighteners, pharmaceuticals, and bacteriostats were manufactured at the Site. By May 1986, Ciba had ceased chemical manufacturing operations at the Site and had begun decommissioning and razing the buildings on Site.

The Site has been divided into three study areas - the Production Area, the Waste Water Treatment Area, and the Warwick Area. The Pawtuxet River (an off-site area) runs through the Site. Twelve solid waste management units (SWMUs) and two areas of concern (AOCs) were identified at the Site. For completeness, Ciba identified two additional areas of investigation (AAOIs). Additional details about the SWMUs, AOCs and AAOIs (and on past known and/or suspected releases were presented in Chapter 1 of the Phase I Interim Report (November 1991). The locations and Media of Concern that were sampled in each of the SWMUs, AOCs, and AAOIs are shown in Figure 1-1.

1.2.2 History of the Project

A draft Administrative Order of Consent (hereafter called the "Order") requiring a

RCRA Corrective Action Study at the Site was issued to Ciba on September 30, 1988. After negotiations and evaluation of public comments, the Order was signed by Ciba on June 9, 1989 and became effective on June 16, 1989. In 1987 USEPA conducted the Facility Assessment to identify known and/or suspected releases at the Site requiring further action. The results were presented in the Final RFA Report, Ciba-Geigy RCRA Facility Assessment (January 1988). Ciba conducted a Preliminary Investigation (not required by the Order) to begin characterizing the Site and selected releases.

1.2.3 History of the Stabilization Investigation

The stabilization investigation was integrated into the RCRA Facility Investigation (RFI) through a Modification of the Order executed on 28 September 1992. The Stabilization Work Plan was submitted to the USEPA in September 1992; conditional approval of the Work Plan was granted on 21 December 1992. The Stabilization Investigation Report and Design Concepts Proposal was submitted to the USEPA in May 1993. The Draft Stabilization Design Documents were submitted to the USEPA in November 1993. The Final Stabilization Design Documents were submitted to the USEPA in June 1994. These final design documents were revised and resubmitted on January 30, 1995.

1.2.4 History of the IRMs

Ciba has elected to move forward with implementing Interim Remedial Measures (IRMs) at the Site. Soil contaminated with PCBs (above the required cleanup levels) in the Production Area and at SWMU-5 will be sampled for waste characteristics, excavated, and landfilled. At SWMU-6, the zinc oxide/soil pile (not a hazardous waste) will be sampled also for waste characteristics, excavated, and landfilled.

Ciba is aware that the remedies proposed in this Work Plan are interim and cannot be approved as the final remedy until the Corrective Measures Study (CMS) is completed. In developing this IRM Work Plan, Ciba and its consultants exercised conservative scientific judgement. The cleanup criteria that have been proposed are risk-based. The risk assessment that was used to develop these criteria is included in Appendix A. Comments generated by USEPA (at our 12/13/94 meeting) on our approach and scope of work were addressed in this Work Plan.

1.3 INTEGRATION WITH STABILIZATION

The IRM proposed for the Production Area will need to be completed prior to conducting the construction activities proposed for stabilization. Specifically, contaminated soil will need to be excavated before the soil vapor extraction system can be installed at SWMU-11 and before a parking lot can be constructed on a

portion of the Production Area. The schedule for implementing the IRM and conducting stabilization will have to be monitored closely to keep both of these tasks on track. This scheduling issue is addressed in more detail in Chapter 8 (Project Management).

1.4 INTEGRATION WITH THE CORRECTIVE MEASURES STUDY

Ciba believes that the IRMs proposed in this Work Plan will be the remedies that will be selected after USEPA reviews the RFI and CMS Reports. It is likely that the IRMs will be implemented prior to the submittal of the CMS Report during the summer of 1995. Scheduling issues are described in more detail in Chapter 8 (Project Management).

1.5 RELATIONSHIP TO PCB REGULATIONS

The USEPA established a cleanup policy for PCBs spilled after May 4, 1987 (40 CFR Part 761, Subpart G). While Ciba can demonstrate that the PCBs found in the Production Area and the Warwick Property soils are the results of pre-1987 "spills," USEPA-Region I has indicated that the policy will apply. However, the policy clearly states that old spoils, discovered after 1987, will be evaluated on a site specific basis and will be cleaned up to requirements "established at the discretion of the USEPA, usually through its regional offices" (40 CFR 761.120(a)(1)(ii)). For the purposes of guidance, the spill policy established soil cleanup levels as follows:

- Non-restricted access areas, such as the Warwick Area, which could be developed for residential or commercial use, 10 ppm total PCBs by weight with clean backfill not to exceed 1 ppm (40 CFR 761.125(c)(3)(v)).
- Restricted access areas, such as the Production Area which is zoned industrial, and would be fenced for parking, 25 ppm total PCBs by weight (40 CFR 761.125(c)(3)(v)).

On December 12, 1994, the USEPA proposed major revisions to the existing PCB regulations (40 CFR 761). A new part specifically addressing PCB remediation waste has been added (40 CFR 761.61(c)). It provides for risk based disposal of PCBs that would be consistent with leaving concentrations above the spill guidance in place. This site specific evaluation is to consider the risk factors associated with the waste and the selected management option, along with applicable USEPA guidelines, criteria, and regulations. The regional USEPA offices again allowed discretion in selecting a cleanup level.

All of the preceding concerns have been addressed in this IRM, especially the risk factors associated with the wastes.

1.6 OBJECTIVES

These IRMs will be performed to meet the following three objectives:

Excavation and disposal of PCB-contaminated soil in Production Area. A cleanup level of 45 ppm will be used for soil in the Production Area. This concentration was determined by taking the preliminary risk-based industrial cleanup level (50 ppm) and subtracting 10 percent to add a level of conservatism. In general, excavation of contaminated soil will be limited to a depth of 1-foot (unless further excavation is required based on post-excavation sampling results). The volume of soil is estimated at 779 cubic yards.

Excavation and disposal of PCB-contaminated soil at SWMU-5.

Because of the sensitivity associated with residential areas, the proposed EPA residential cleanup level (1 ppm) will be targeted in SWMU-5, rather than the preliminary risk-based residential level (9 ppm). Soil contaminated with more than 1 ppm PCBs will be excavated to a depth of 2-feet. The volume of soil is estimated at 210 cubic yards.

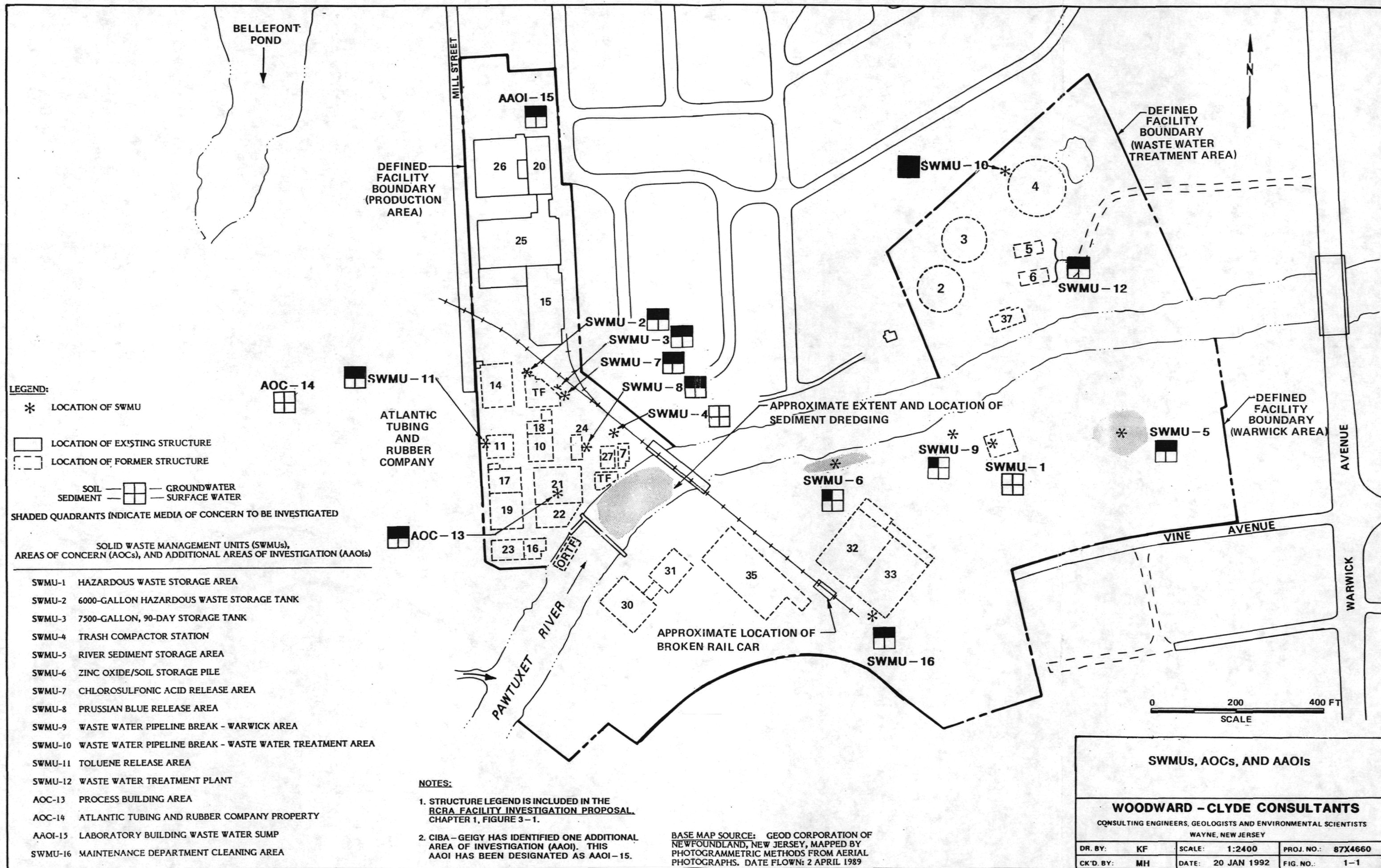
Excavation and disposal of the zinc oxide/soil pile at SWMU-6. The zinc oxide/soil pile, currently staged on asphalt, will be removed and disposed. The volume of soil is estimated at 30 cubic yards.

1.7 ORGANIZATION

This report has seven additional chapters:

- Chapter 2 describes the risk assessment and the Media Protection Standards.
- Chapter 3 briefly reviews the existing analytical data for the Production Area, and SWMU-5 and SWMU-6 in the Warwick Area.
- Chapter 4 describes the tasks to be completed before the preparation of the bid specifications and implementation of the field program.
- Chapter 5 describes the field program for the Production Area.
- Chapter 6 describes the field program for SWMU-5.
- Chapter 7 describes the field program for SWMU-6.

- Chapter 8 discusses the management of the project during the soil excavation IRM.



RISK ASSESSMENT AND MEDIA PROTECTION STANDARDS

This Risk Assessment was prepared to support the IRMs in the Production and Warwick Areas proposed by Ciba for the Site. This chapter is a brief summary of the comprehensive Risk Assessment provided in its entirety in Appendix A. It separately evaluates the potential human health risks associated with the Production and Warwick Areas. It is consistent with the approach outlined in the USEPA's primary risk assessment guidance documents. The Risk Assessment approach and values for exposure assumptions reflect discussions held with the USEPA Region I (Region I) during several meetings and teleconferences, beginning with the May 17, 1994, meeting with Ciba at the Region I offices.

The purpose of the Risk Assessment is threefold:

- Provide estimates of potential risks posed by site-related chemicals in the Production and Warwick Areas of the Site using the conservative guidance specified by Region I.
- Identify the site areas and chemicals that might require corrective action using this risk assessment approach.
- Provide a site-specific risk assessment model using this conservative approach for estimating risk-based Media Protection Standards (MPS) for surface soil.

The Risk Assessment is designed to provide a conservative, quantitative estimate of potential risks associated with residual site-related chemicals in the Production and Warwick Areas. It is based on analytical results from soil samples collected during Phase I and II of the RCRA Facility Investigation field activities. It was performed by identifying chemicals of potential concern (COPC) and carrying them through the risk assessment process. The COPC were determined based on their toxicities, frequencies of detection, and concentrations in site soil.

Regarding future land use, separate exposure scenarios were evaluated for the Production and Warwick Areas. Based on a proposal to use the Production Area as a vehicle parking facility, the Risk Assessment reflects an on-site worker scenario for this area. Unrestricted residential land use was assumed for the Warwick Area.

Results of the Risk Assessment are expressed in terms of potential noncancer health effects and potential cancer risks which are summarized in Figures 2-1 and 2-2. The total hazard index (THI) represents the overall estimated noncancer risks for a given exposure scenario. The potential noncancer risk represented by the THI is considered of no significance if it is equal to

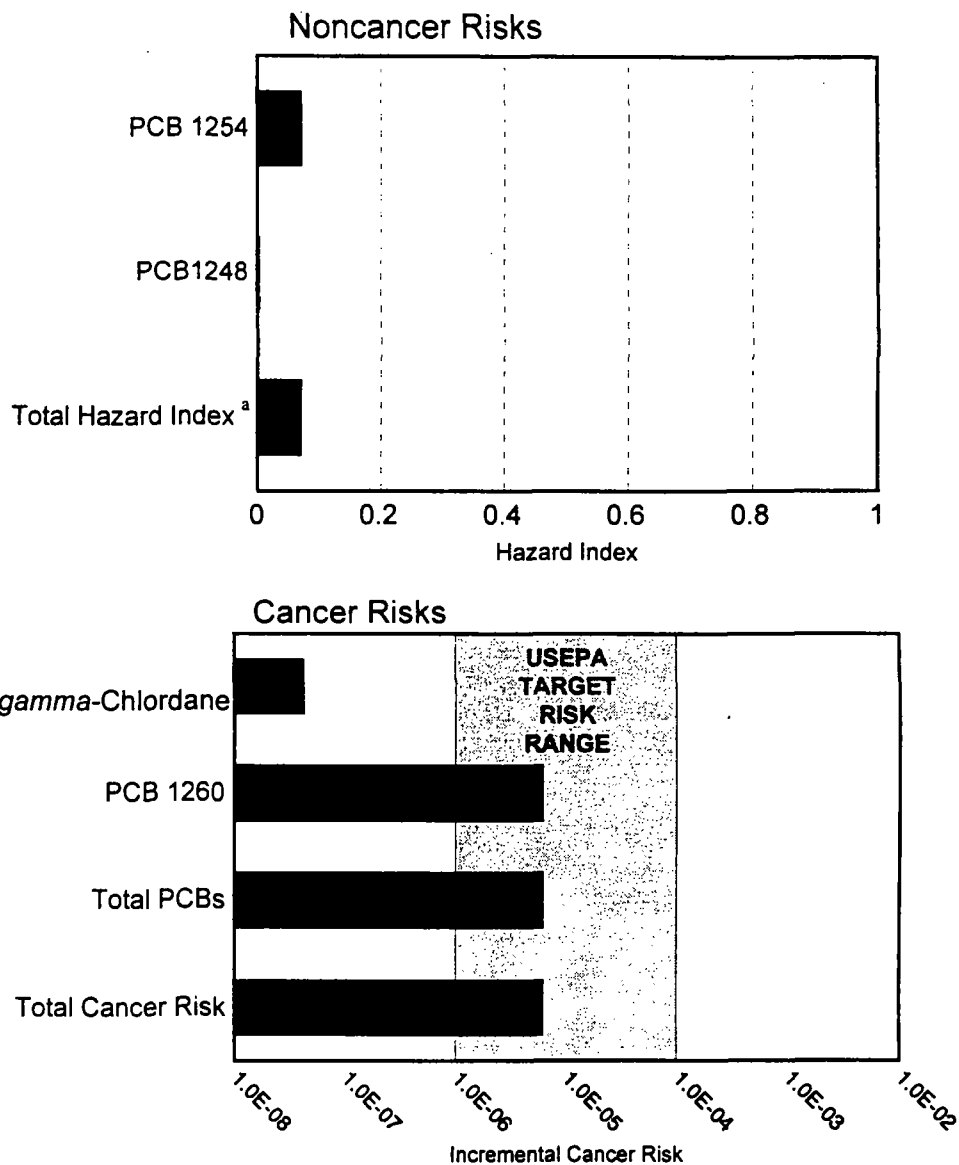
or below a value of 1, and is a potential concern if it is greater than a value of 1 (rounded to a whole number). The potential cancer risk posed is expressed in terms of an incremental lifetime cancer risk (ILCR). The ILCR is an increased probability of cancer above that which exists as "background" (3 out of 10 people) for the general population. The USEPA regards an ILCR of between 1×10^{-6} (1 in 1,000,000) and 1×10^{-4} (1 in 10,000) as acceptable. Thus, this may be interpreted as an increase in the United States baseline cancer incidence from 300,000 per million population to a range of 300,001 to 300,100 per million population. If the ILCR exceeds the upper bound of the target risk range (1×10^{-4}), then further evaluation or corrective action may be indicated.

As shown in Figures 2-1 and 2-2, neither the Production Area nor the Warwick Area are predicted to pose an unacceptable potential risk. The risk numbers presented are highly conservative and may exaggerate actual risks due to a number of factors. For example, the sampling approach was biased in that the field investigation targeted highly localized areas of suspected contamination. Additionally, at Region I's request, the total PCB carcinogenic risk is based on the assumption that all PCBs, including those that are noncarcinogenic (e.g. PCB 1248 and 1254) have a cancer potency factor equal to PCB 1260. These factors are especially significant for the Warwick area, where contamination (PCB 1248 and 1254) is highly localized and no PCB 1260 was detected. From a land use standpoint, the likelihood of PCB exposure through surface soil is highly unlikely in the Production Area, since the proposed land use is a paved parking facility.

Even with the high degree of conservatism, the Risk Assessment showed that corrective actions are not necessary for the Production and Warwick areas solely on the basis of potential risk to public health. However, it may be desirable to conduct some limited remediation in these areas for reasons other than potential risk, such as facilitating the productive use of these areas. Based on the concentration and frequency of detection in surface soil (the predominant exposure source), it was determined that PCB removal in the Production and Warwick Areas would provide the greatest benefit in potential risk reduction. Therefore, proposed surface soil MPS values are limited to PCBs only.

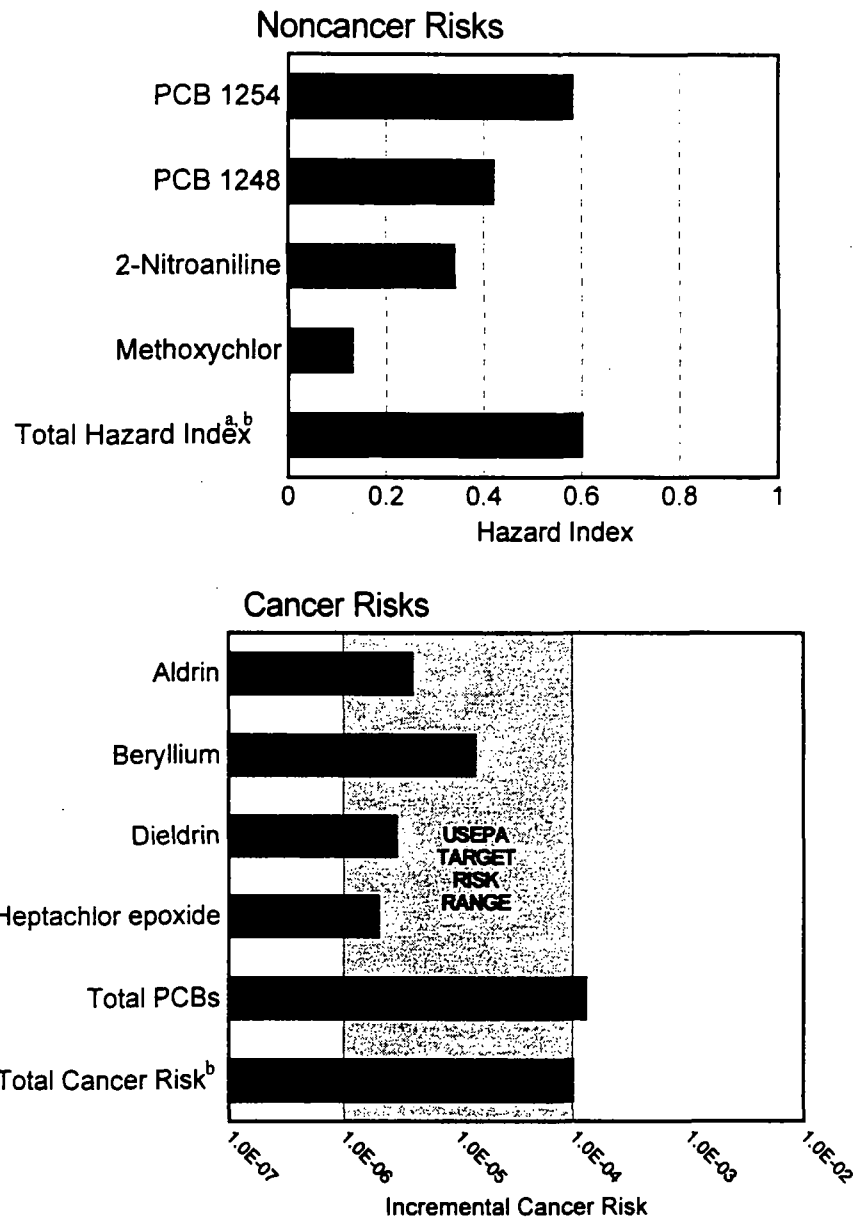
The risk assessment models for the scenarios evaluated were used to estimate risk-based MPS values for total PCBs. Using a THI value of 1, MPSs were back-calculated through the risk assessment model to the respective surface soil concentrations. The resulting total PCBs MPSs are 50 ppm for the Production Area and 9 ppm for the Warwick Area. A clean-up level of 45 ppm (5 ppm lower than that allowed by the risk-based MPS) will be targeted for the Production Area to ensure that the average residual PCB concentration is below the 50 ppm limit. Based on draft USEPA guidance (Disposal of Polychlorinated Biphenyls;

Proposed Rule 12/12/94), the decision was made to reduce the target clean-up level in the Warwick Area to 1 ppm to allow for unrestricted use.



^a Only similar hazards are summed. Refer to Appendix A, Section A7.0.

Figure 2-1. Risk Summary for Production Area On-Site Worker Scenario



^a Only similar hazards are summed. Refer to Appendix A, Section A7.0.

^b Rounded to one significant figure, as recommended by the USEPA.

Figure 2-2. Risk Summary for Warwick Area On-Site Resident Scenario

REVIEW OF EXISTING DATA

3.1 OVERVIEW

This chapter presents the results from the analysis of soil from the Production Area, at SWMU-5 (River Sediment Storage Area), and at SWMU-6 (Zinc Oxide/Soil Pile). The discussion is limited to PCBs (in the Production Area and at SWMU-5) and zinc (at SWMU-6). Other organic or inorganic constituent results will not be reviewed here because the focus of the IRMs is limited to the aforementioned analytes. These additional results will be presented in the RFI Report.

The data presented here include the results of sampling conducted in Phases I and II of the RFI. Total PCBs were calculated using the sum of Aroclors 1248, 1254 and 1260. If the compound was not detected, one half of the detection limit was included in the total. Analytical laboratory PCB methodology including QA/QC and WCCs data validation procedures are presented in Appendix B. The analytical results from preliminary waste classification sampling in the Production Area and SWMU-5 are presented in Appendix C.

3.2 PRODUCTION AREA

A total of 142 soil samples (not including field duplicates) collected in the Production Area were analyzed for Appendix IX PCBs (Tables 3-1 through 3-3). An additional 18 soil samples also were collected and analyzed for engineering grade PCBs. These samples were collected at depths ranging from 0.5 to 10 ft below ground surface. Samples from the 0.5 to 1.0-ft interval were collected manually. All other samples, including the samples from the 0-2 ft interval, were collected using split-spoon samplers during the advancement of soil borings. All surface soil sampling locations (0.5 to 1.0 ft) and soil boring locations are shown on Figure 3-1.

The analytical data show that none of the samples collected at depths greater than 2 ft below ground surface contained PCBs in concentrations which exceed the IRM cleanup level of 45 ppm (Table 3-1).

One sample collected from the 0 to 2 ft-interval contained PCBs (4,900 ppm) at a concentration exceeding the IRM cleanup level (Table 3-2).

Thirteen (not including two field duplicates) of the 71 samples collected from the 0.5 to 1 ft-interval contained PCBs in concentrations greater than 45 ppm (Table 3-3). Figure 3-1 shows the estimated area of soil in the Production Area containing PCBs in concentrations exceeding the IRM cleanup level.

On December 27, 1994 soil was sampled at three locations in the Production Area. Sample locations were biased towards areas where previous sampling events showed elevated levels of PCBs. These samples were submitted to Ciba's Environmental Testing Laboratory (CETL) in Toms River, New Jersey to be analyzed for TCLP/RCRA Characteristics. The results of these analyses (Appendix C) indicate that these samples were RCRA non-hazardous.

3.3 SWMU-5: RIVER SEDIMENT STORAGE AREA

SWMU-5 was a storage area for sediment dredged from the Pawtuxet River. 6,630 cubic yards of material were removed from the storage area in 1976 as part of a flood plain restoration program. The exact limits of the stockpile are not known.

Twenty-nine samples (not including one field duplicate) were collected from various depths in the SWMU-5 area during Phases I and II of the RFI. Of these, one sample was rejected during data validation. Figure 3-2 shows the locations where these samples were collected. This figure also presents detected results (shown in yellow) and total PCB concentrations (shown in blue) for these samples.

Concentrations of PCBs in six (not counting one field duplicate) of the samples exceeded the USEPA cleanup level for residential sites of 1 ppm (Table 3-4). This does not include samples for which detection limits, rather than actual concentrations are driving the exceedance. All of the actual exceedances are in samples collected from the 0 to 2-ft interval. If the area of soil estimated to exceed 1 ppm PCBs is extracted from the existing data points, the concentration of PCBs in one sample (SF-S5-ZZ3(D)*IB-2 : 160 ppm) and detection limits for other samples cause the extrapolated area to extend roughly to the outer most samples. This approach is probably an overly conservative estimation. Therefore, the limits of excavation were determined by visually delineating the area where actual PCB concentrations exceeded 1 ppm. Post-excavation analytical results will be used to determine if this approach was reasonable. In addition, previous release characterization sampling locations for which detection limits exceeded the cleanup level will be resampled, prior to excavating soils in SWMU-5, to verify the assumption that PCBs, if present, are below 1 ppm. Figure 3-2 shows the estimated area of soil in SWMU-5 containing PCBs in concentrations exceeding the 1 ppm cleanup level based on the assumptions described above.

On December 27, 1994 soil was sampled at seven locations in SWMU-5. Sample locations were biased towards areas where previous sampling events showed elevated levels of PCBs. These samples were submitted to Ciba's Environmental Testing Laboratory (CETL) in Toms River, New Jersey to be analyzed for TCLP/RCRA Characteristics. The results of these analyses (Appendix C) indicate that these samples were RCRA non-hazardous.

3.4 SWMU-6: ZINC OXIDE/SOIL PILE

SWMU-6 is a soil pile containing residues of zinc oxide from a railcar spill in the late 1960s. Road sweepings from in and around the spill were used to form a drainage berm now identified as SWMU-6. The berm, approximately 50 ft long by 7 ft wide by 2 ft high, contains approximately 25-30 cubic yards of material. The bulk of the berm is staged on an asphalted surface.

Four surface soil samples were collected from the SWMU-6 area, two within the stockpile and two from the potentially impacted soil between the stockpile and the Pawtuxet River. The samples within the stockpile had zinc concentrations of 850 ppm and 2390 ppm (Table 3-5). Samples from the potentially impacted soil had zinc concentrations of 111 ppm and 56.7 ppm (within the limits of background soil concentrations for this region).

TABLE 3-1
PRODUCTION AREA
PCBs IN SOIL BORING SAMPLES (greater than 2 ft)

PHASE / ROUND	IB-1	IB-2	II-1	II-1	II-1	II-1	II-1	II-1	II-1	II-1	IB-2	IB-2	II-1													
SUB AREA / LOCATION	SMU7/B7A	SMU3/B3C	SMU2/B2G2	SMU3/B3E2	SMU3/B3G2	SMU3/B3H2	SMU7/B7H2	SMU8/B8E2	SMU8/B8G2	SMU8/B8H2	SMU7/B7C	SMU8/B8C	AOC13/B13A3													
SAMPLE ID	B-7A*IB-1	B-3C*IB-2	B-2G2*II-1	B-3E2*II-1	B-3G2*II-1	B-3H2*II-1	B-7H2*II-1	B-8E2*II-1	B-8G2*II-1	B-8H2*II-1	B-7C*IB-2	B-8C*IB-2	B-13A3*II-1													
DEPTH FROM (FT)	2	2	2	2	2	2	2	2	2	2	4	4	4													
DEPTH TO (FT)	4	4	4	4	4	4	4	4	4	4	6	6	6													
COLLECT DATE	11/20/90	3/18/91	7/9/93	7/12/93	7/12/93	7/12/93	7/23/93	7/24/93	7/24/93	7/24/93	3/18/91	3/14/91	7/20/93													
	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q												
PCB-1016	1 U		0.11 U		0.18 U		0.034 U		0.035 U		0.37 U		0.034 U		0.17 U		0.035 U		0.011 U		0.12 U		0.035 U			
PCB-1221	2 U		0.22 U		0.36 U		0.07 U		0.071 U		0.18 U		0.068 U		0.35 U		0.071 U		0.022 U		0.23 U		0.072 U			
PCB-1232	2 U		0.22 U		0.18 U		0.034 U		0.035 U		0.18 U		0.034 U		0.17 U		0.035 U		0.022 U		0.23 U		0.035 U			
PCB-1242	1 U		0.11 U		0.18 U		0.034 U		0.035 U		0.18 U		0.034 U		0.17 U		0.035 U		0.011 U		0.12 U		0.035 U			
PCB-1248	1 U		0.11 U		0.18 U		0.034 U		0.035 U		0.18 U		0.034 U		0.17 U		0.035 U		0.011 U		0.12 U		0.035 U			
PCB-1254	2 U		1.4		2.4		0.44		1.2 J		3		1.6 J		0.26		0.95		0.21		0.51		3.5 J		0.34	
PCB-1260	13 J		0.22 U		0.18 U		1.81 D		0.035 U		0.035 U		0.18 U		0.034 U		0.17 U		0.035 U		0.022 U		0.23 U		0.035 U	
	II-1	II-1	II-1	II-1	II-1	II-1	II-1	II-1	II-1	II-1	IB-1	IB-1	IB-1	IB-1	IB-1	IB-1	IB-2	IB-2								
	SMU2/B2E3	SMU2/B2G3	SMU3/B3I3	SMU7/B7F3	SMU8/B8D3	SMU8/B8E3	SMU8/B8F3	SMU8/B8F3	SMU2/B2B	SMU3/B3A	SMU3/B3B	SMU2/B2B	SMU2/B2C													
	B-2E3*II-1	B-2G3*II-1	B-3I3*II-1	B-7F3*II-1	B-8D3*II-1	B-8E3*II-1	B-8F3*II-1	B-DUP3*II-1	B-2B*IB-1	B-3A*IB-1	B-3B*IB-1	B-2B*IB-2	B-2C*IB-2													
	4	4	4	4	4	4	4	4	6	6	6	6	6													
	6	6	6	6	6	6	6	6	8	8	8	8	8													
	7/9/93	7/9/93	7/13/93	7/23/93	7/24/93	7/24/93	7/24/93	7/24/93	12/6/90	11/20/90	11/19/90	3/14/91	3/14/91													
	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q		
PCB-1016	0.035 U		0.034 U		0.035 U		0.72 U		0.036 U		0.035 U		0.04 U		0.038 U		0.011 U		0.11 U		0.11 U		0.011 U		0.052 U	
PCB-1221	0.07 U		0.07 U		0.071 U		1.5 U		0.073 U		0.07 U		0.081 U		0.077 U		0.023 U		0.22 U		0.21 U		0.022 U		0.1 U	
PCB-1232	0.035 U		0.034 U		0.035 U		0.72 U		0.036 U		0.035 U		0.04 U		0.038 U		0.023 U		0.22 U		0.21 U		0.022 U		0.1 U	
PCB-1242	0.035 U		0.034 U		0.035 U		0.72 U		0.036 U		0.035 U		0.04 U		0.038 U		0.011 U		0.11 U		0.11 U		0.011 U		0.052 U	
PCB-1248	0.035 U		0.034 U		0.035 U		0.72 U		0.036 U		0.035 U		0.04 U		0.038 U		0.011 U		0.11 U		0.11 U		0.011 U		0.052 U	
PCB-1254	0.12		0.034 U		0.2 J		0.72 U		0.058		0.035 U		0.11		0.1		0.85 J		0.22 U		3.8		0.21		0.78	
PCB-1260	0.035 U		0.034 U		0.035 U		0.72 U		0.036 U		0.035 U		0.04 U		0.038 U		0.023 U		3.3		0.21 U		0.022 U		0.1 U	
	IB-2	IB-2	IB-2	II-1	II-1	II-1	II-1	II-1	II-1	II-1	II-1	II-1	IB-1													
	SMU2/B2D	SMU3/B3A	SMU3/B3D	AOC13/B13A4	SMU2/B2F4	SMU3/B3E4	SMU3/B3F4	SMU3/B3H4	SMU7/B7D4	SMU7/B7E4	SMU7/B7G4	SMU7/B7H4	SMU11/B11A													
	B-2D*IB-2	B-3A*IB-2	B-3D*IB-2	B-13A4*II-1	B-2F4*II-1	B-3E4*II-1	B-3F4*II-1	B-3H4*II-1	B-7D4*II-1	B-7E4*II-1	B-7G4*II-1	B-7H4*II-1	B-11A*IB-1													
	6	6	6	6	6	6	6	6	6	6	6	6	3													
	8	8	8	8	8	8	8	8	8	8	8	8	5													
	3/15/91	3/18/91	3/18/91	7/20/93	7/9/93	7/12/93	7/12/93	7/12/93	7/23/93	7/23/93	7/23/93	7/23/93	12/6/90													
	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q		
PCB-1016	0.011 U		0.11 U		0.11 U		0.034 U		0.035 U		0.18 U		0.035 U		0.035 U		0.18 U		0.17 U		0.036 U		0.034 U		0.1 U	
PCB-1221	0.021 U		0.22 U		0.22 U		0.07 U		0.072 U		0.37 U		0.071 U		0.07 U		0.36 U		0.35 U		0.073 U		0.07 U		0.21 U	
PCB-1232	0.021 U		0.22 U		0.22 U		0.034 U		0.035 U		0.18 U		0.035 U		0.035 U		0.18 U		0.17 U		0.036 U		0.034 U		0.21 U	
PCB-1242	0.011 U		0.11 U		0.11 U		0.034 U		0.035 U		0.18 U		0.035 U		0.035 U		0.18 U		0.17 U		0.036 U		0.034 U		0.1 U	
PCB-1248	0.011 U		0.11 U		0.11 U		0.034 U		0.035 U		0.18 U		0.035 U		0.035 U		0.18 U		0.17 U		0.036 U		0.034 U		0.1 U	
PCB-1254	0.19 J		4.3		3.4		0.074		0.054		0.95		0.054		0.54		0.92		0.17 U		0.27 J		0.36		0.21 U	
PCB-1260	0.021 U		0.22 U		0.22 U		0.034 U		0.035 U		2.6		0.07		0.64		1.9		2.6		0.036 U		0.034 U		0.21 U	
	IB-1	IB-1	IB-2	IB-2																						
	SMU11/B11B	SMU2/B2A	SMU11/B11B	SMU11/B11C																						
	B-11B*IB-1	B-2A*IB-1	B-11B*IB-2	B-11C*IB-2																						
	3	8	3	5																						
	7	10	7	7																						
	11/20/90	11/19/90	3/15/91	3/15/91																						
	Result	Q	Result	Q	Result	Q	Result	Q																		
PCB-1016	1 U		0.013 U		0.52 U		0.51 U																			
PCB-1221	2.1 U		0.025 U		1 U		1 U																			
PCB-1232	2.1 U		0.025 U		1 U		1 U																			
PCB-1242	1 U		0.013 U		0.52 U		0.51 U																			
PCB-1248	1 U		0.013 U		0.52 U		0.51 U																			
PCB-1254	2.1 U		0.025 U		9		1 U																			
PCB-1260	2.1 U		0.025 U		1 U		1 U																			

All results reported in mg/kg (ppm)

All nondetected results reported at full detection limits

U - Undetected

J - Estimated result

D - Diluted sample

R - Rejected result

All results reported in mg/kg (ppm)
All nondetected results reported at full detection limits
U - Undetected
J - Estimated result
D - Diluted sample
R - Rejected result

TABLE 3-2
PRODUCTION AREA
PCB'S IN SOILBORING SAMPLES (0 TO 2 FT)

1/31/95

PHASE / ROUND	IR-1	IR-1	IR-1	IR-2	IR-2	II-1	II-1	II-1	II-1	II-1	II-1	II-1	II-1													
SUR AREA / LOCATION	SMU7/B7R	SMU8/B8A	SMU8/B8R	SMU7/B7R	SMU8/B8R	SMU2/B2E1	SMU2/B2F1	SMU3/B3E1	SMU3/B3F1	SMU3/B3G1	SMU3/B3I1	SMU7/R7D1	SMU7/R7D1													
SAMPLE ID	R-7B*IR-1	B-8A*IR-1	R-8B*IR-1	B-7B*IR-2	B-8B*IR-2	R-2E1*II-1	R-2F1*II-1	R-3E1*II-1	B-3F1*II-1	B-3G1*II-1	B-3I1*II-1	R-7D1*II-1	R-DUP2*II-1													
DEPTH FROM (FT)	0	0	0	0	0	0	0	0	0	0	0	0	0													
DEPTH TO (FT)	2	2	2	2	2	2	2	2	2	2	2	2	2													
COLLECT DATE	11/20/90	11/20/90	11/20/90	3/18/91	3/14/91	7/9/93	7/9/93	7/12/93	7/12/93	7/12/93	7/13/93	7/23/93	7/23/93													
	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q												
PCB-1016	0.1 U		0.052 U		0.53 U		0.55 U		0.34 U		0.34 U		0.034 U		0.17 U		0.67 U		0.34 U							
PCB-1221	0.21 U		0.1 U		1.1 U		1.1 U		0.068 U		0.071 U		0.68 U		0.68 U		0.069 U		0.34 U		1.4 U		0.68 U			
PCB-1232	0.21 U		0.1 U		1.1 U		1.1 U		0.34 U		0.035 U		0.34 U		0.34 U		0.034 U		0.17 U		0.67 U		0.34 U			
PCB-1242	0.1 U		0.052 U		0.53 U		0.55 U		0.34 U		0.035 U		0.34 U		0.34 U		0.034 U		0.17 U		0.67 U		0.34 U			
PCB-1248	0.1 U		0.052 U		0.53 U		0.55 U		0.56 U		0.034 U		0.035 U		0.34 U		0.034 U		0.17 U		0.67 U		0.34 U			
PCB-1254	5.2		1.8		1.1 U		6		12 J		0.15 J		0.19		2.3		5.3		0.99 J		4.8 J		2.2 J		1.4	
PCB-1260	0.21 U		0.1 U		1.1 U		1.1 U		0.13		0.24		3.2		3		0.034 U		0.17 U		6.1 J		2.7 J			

	II-1	II-1	II-1	II-1	II-1	II-1	II-1	II-1	II-1	II-2	II-2	II-2	II-2	II-2												
	SMU7/B7E1	SMU7/B7F1	SMU7/B7G1	SMU7/B7H1	SMU8/B8D1	SMU8/B8F1	SMU8/B8G1	SMU8/B8H1	SMU8/B8I1	AOC-13/AD152	AOC-13/E162	AOC-13/F362	AOC-13/F452	AOC-13/O162												
	B-7E1*II-1	B-7F1*II-1	B-7G1*II-1	B-7H1*II-1	B-8D1*II-1	B-8F1*II-1	B-8G1*II-1	B-8H1*II-1	B-8I1*II-1	B-13AD152*II-2	B-13E162*II-2	B-13F362*II-2	B-13F452*II-2	B-13O162*II-2												
	0	0	0	0	0	0	0	0	0	0	0	0	0	0												
	2	2	2	2	2	2	2	2	2	2	2	2	2	2												
	7/23/93	7/23/93	7/23/93	7/23/93	7/24/93	7/24/93	7/24/93	7/24/93	7/24/93	5/4/94	5/4/94	5/4/94	5/4/94	5/4/94												
	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q								
PCB-1016	0.17 U		0.034 U		0.17 U		0.35 U		0.034 U		0.17 U		0.7 U		0.035 U		0.036 U		0.18 U		0.37 U		0.71 U		0.069 U	
PCB-1221	0.35 U		0.069 U		0.35 U		0.72 U		0.07 U		0.35 U		1.4 U		0.07 U		0.072 U		0.38 U		0.75 U		1.4 U		0.14 U	
PCB-1232	0.17 U		0.034 U		0.17 U		0.35 U		0.034 U		0.17 U		0.7 U		0.035 U		0.036 U		0.18 U		0.37 U		0.71 U		0.069 U	
PCB-1242	0.17 U		0.034 U		0.17 U		0.35 U		0.034 U		0.17 U		0.7 U		0.035 U		0.036 U		0.18 U		0.37 U		0.71 U		0.069 U	
PCB-1248	0.17 U		0.034 U		0.17 U		0.35 U		0.034 U		0.17 U		0.7 U		0.035 U		0.036 U		0.18 U		0.37 U		0.71 U		0.069 U	
PCB-1254	0.62		0.034 U		2.2		4.2 J		0.052		1.1		7.8		0.4 J		0.036 U		0.18 U		1.3 J		6.1		0.55	
PCB-1260	0.83		0.034 U		0.17 U		0.35 U		0.034 U		0.17 U		0.7 U		0.035 U		0.036 U		0.18 U		0.37 U		0.71 U		0.069 U	

	II-2	II-2	II-2	II-2				
	AOC-13/O262	AOC-13/O362	AOC-13/O452	AOC-13/Y262				
	B-13O262*II-2	B-13O362*II-2	B-13O452*II-2	B-13Y262*II-2				
	0	0	0	0				
	2	2	2	2				
	5/4/94	5/4/94	5/4/94	5/4/94				
	Result	Q	Result	Q	Result	Q	Result	Q
PCB-1016		0.35 U		0.34 U		400 U		0.37 U
PCB-1221		0.71 U		0.69 U		820 U		0.75 U
PCB-1232		0.35 U		0.34 U		400 U		0.37 U
PCB-1242		0.35 U		0.34 U		400 U		0.37 U
PCB-1248		0.35 U		3.6		4500		0.37 U
PCB-1254		0.4 J		0.34 U		400 U		1.2 J
PCB-1260		0.35 U		0.34 U		400 U		0.37 U

All results reported in mg/kg (ppm).
All nondetected results reported at full detection limits.
U - undetected.
J - Estimated result.
D - Diluted sample.
R - Rejected result.

TABLE 3-3
PRODUCTION AREA
PCB'S IN SURFACE SOIL SAMPLES (0.5 - 1 FT)

PHASE / ROUND SUB AREA / LOCATION SAMPLE ID	IB-1 AOC13/A25 SF-A13-A25(S)*IB-1	IB-1 AOC13/A40 SF-A13-A40(S)*IB-1	IB-1 AOC13/E45 SF-A13-E45(S)*IB-1	IB-1 AOC13/J30 SF-A13-J30(S)*IB-1	IB-1 AOC13/J35 SF-A13-J35(S)*IB-1	IB-1 AOC13/J40 SF-A13-J40(S)*IB-1	IB-1 AOC13/O10 SF-A13-O10(S)*IB-1	IB-1 AOC13/O25 SF-A13-O25(S)*IB-1	IB-1 AOC13/T10 SF-A13-T10(S)*IB-1	IB-1 AOC13/Y5 SF-A13-Y5(S)*IB-1	IB-1 AOC13/Y5 SF-DUP-1*IB-1	IB-2 AOC13/AA7 SF-A13-AA7(S)*IB-2	IB-2 AOC13/C27 SF-A13-C27(S)*IB-2	IB-2 AOC13/C41 SF-A13-C41(S)*IB-2	IB-2 AOC13/G47 SF-A13-G47(S)*IB-2	IB-2 AOC13/J40 SF-A13-J40(S)*IB-2
DEPTH FROM (FT)	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
DEPTH TO (FT)	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
COLLECT DATE	11/15/90	11/14/90	11/14/90	11/14/90	11/14/90	11/14/90	11/14/90	12/6/90	11/14/90	11/14/90	11/14/90	3/15/91	3/14/91	3/14/91	3/14/91	3/14/91
	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q
PCB-1016	1.3 U	1.1 U	1.1 U	1.1 U	1.2 U	1.1 U	0.11 U	0.011 U	0.1 U	0.01 U	0.01 U	0.011 U	2.3 U	0.55 U	0.53 U	2.3 U
PCB-1221	2.6 U	2.3 U	2.1 U	2.2 U	2.3 U	2.2 U	0.22 U	0.022 U	0.21 U	0.02 U	0.02 U	0.021 U	4.5 U	1.1 U	1.1 U	4.5 U
PCB-1232	2.6 U	2.3 U	2.1 U	2.2 U	2.3 U	2.2 U	0.22 U	0.022 U	0.21 U	0.02 U	0.02 U	0.021 U	4.5 U	1.1 U	1.1 U	4.5 U
PCB-1242	1.3 U	1.1 U	1.1 U	1.1 U	1.2 U	1.1 U	0.11 U	0.011 U	0.1 U	0.01 U	0.01 U	0.011 U	2.3 U	0.55 U	0.53 U	2.3 U
PCB-1248	1.3 U	1.1 U	1.1 U	1.1 U	1.2 U	1.1 U	0.11 U	0.011 U	0.1 U	0.01 U	0.01 U	0.011 U	2.3 U	0.55 U	0.53 U	2.3 U
PCB-1254	29	25	51	22	37	51	4	1.4 J	2.7	0.02 U	0.02 U	0.099	75	14	6.5	77
PCB-1260	2.6 U	2.3 U	2.1 U	2.2 U	2.3 U	2.2 U	0.22 U	0.022 U	0.21 U	0.02 U	0.02 U	0.021 U	4.5 U	1.1 U	1.1 U	4.5 U
	IB-2 AOC13/L32 SF-A13-L32(S)*IB-2	IB-2 AOC13/L37 SF-A13-L37(S)*IB-2	IB-2 AOC13/O10 SF-A13-O10(S)*IB-2	IB-2 AOC13/Q27 SF-A13-Q27(S)*IB-2	IB-2 AOC13/T10 SF-A13-T10(S)*IB-2	II-1 AOC13/AB21 SS-AB21*II-1	II-1 AOC13/AB24 SS-AB24*II-1	II-1 AOC13/AE11 SS-AE11*II-1	II-1 AOC13/AF26 SS-AF26*II-1	II-1 AOC13/AG23 SS-AG23*II-1	II-1 AOC13/AJ15 SS-AJ15*II-1	II-1 AOC13/B2 SS-B2*II-1	II-1 AOC13/B7 SS-B7*II-1	II-1 AOC13/C16 SS-C16*II-1	II-1 AOC13/C20 SS-C20*II-1	II-1 AOC13/D37 SS-D37*II-1
	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
	3/15/91	3/15/91	3/14/91	3/15/91	3/14/91	4/8/92	4/8/92	4/8/92	4/8/92	4/8/92	4/8/92	4/7/92	4/7/92	4/8/92	4/8/92	4/7/92
	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q
PCB-1016	0.55 U	0.11 U	0.1 U	1.1 U	0.11 U	R	0.037 U	0.034 U	0.17 U	R	0.035 U	0.038 U	0.037 U	R	0.037 U	R
PCB-1221	1.1 U	0.21 U	0.21 U	2.3 U	0.22 U	R	0.074 U	0.07 U	0.35 U	R	0.071 U	0.074 U	R	0.074 U	R	R
PCB-1232	1.1 U	0.21 U	0.21 U	2.3 U	0.22 U	R	0.037 U	0.034 U	0.17 U	R	0.035 U	0.038 U	0.037 U	R	0.037 U	R
PCB-1242	0.55 U	0.11 U	0.1 U	1.1 U	0.11 U	R	0.037 U	0.034 U	0.17 U	R	0.035 U	0.038 U	0.037 U	R	0.037 U	R
PCB-1248	0.55 U	0.11 U	0.1 U	1.1 U	0.11 U	R	0.23	0.034 U	0.37	2 J	0.072	0.35	0.051	15 J	0.12	5.5 J
PCB-1254	22	4.7	5.3	30	1.8	25 J	0.4	0.31	5.2	19 J	0.86	1.7	0.34	84 J	0.6	64 J
PCB-1260	1.1 U	0.21 U	0.21 U	2.3 U	0.22 U	R	0.037 U	0.034 U	0.17 U	R	0.035 U	0.038 U	0.037 U	R	0.037 U	R
	II-1 AOC13/E23 SS-E23*II-1	II-1 AOC13/E31 SS-E31*II-1	II-1 AOC13/E35 SS-E35*II-1	II-1 AOC13/F26 SS-F26*II-1	II-1 AOC13/G38 SS-G38*II-1	II-1 AOC13/I43 SS-I43*II-1	II-1 AOC13/J11 SS-J11*II-1	II-1 AOC13/J21 SS-J21*II-1	II-1 AOC13/J45 SS-J45*II-1	II-1 AOC13/J45 SS-J45*II-1	II-1 AOC13/K14 SS-K14*II-1	II-1 AOC13/K26 SS-K26*II-1	II-1 AOC13/L1 SS-L1*II-1	II-1 AOC13/L16 SS-L16*II-1	II-1 AOC13/L48 SS-L48*II-1	II-1 AOC13/M22 SS-M22*II-1
	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
	4/7/92	4/7/92	4/7/92	4/7/92	4/7/92	4/6/92	4/6/92	4/6/92	4/6/92	4/6/92	4/6/92	4/7/92	4/6/92	4/7/92	4/6/92	4/7/92
	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q
PCB-1016	0.75 U	R	R	R	R	R	0.35 U	0.035 U	R	R	0.19 U	0.17 U	0.35 U	0.18 U	0.035 U	R
PCB-1221	1.5 U	R	R	R	R	R	0.72 U	0.071 U	R	R	0.38 U	0.35 U	0.71 U	0.36 U	0.071 U	R
PCB-1232	0.75 U	R	R	R	R	R	0.35 U	0.035 U	R	R	0.19 U	0.17 U	0.35 U	0.18 U	0.035 U	R
PCB-1242	0.75 U	R	R	R	R	R	0.35 U	0.035 U	R	R	0.19 U	0.17 U	0.35 U	0.18 U	0.035 U	R
PCB-1248	1.3	6.4 J	5.2 J	4.8 J	5.3 J	2 J	0.35 U	0.22 J	19 J	19 J	0.42	0.17 U	0.35 U	0.3	0.32	R
PCB-1254	27	47 J	58 J	74 J	36 J	10 J	4.5	0.5	30 J	32 J	1.4	3.8	4.3 J	0.75	0.64	37 J
PCB-1260	0.75 U	R	R	R	R	R	0.35 U	0.035 U	R	R	0.19 U	0.17 U	0.35 U	0.18 U	0.035 U	R
	II-1 AOC13/M42 SS-M42*II-1	II-1 AOC13/N13 SS-DUP2*II-1	II-1 AOC13/N13 SS-N13*II-1	II-1 AOC13/N29 SS-N29*II-1	II-1 AOC13/N35 SS-N35*II-1	II-1 AOC13/O17 SS-O17*II-1	II-1 AOC13/O44 SF-O44*II-1	II-1 AOC13/O44 SS-O44*II-1	II-1 AOC13/O7 SS-O7*II-1	II-1 AOC13/Q22 SS-Q22*II-1	II-1 AOC13/Q38 SS-Q38*II-1	II-1 AOC13/Q42 SS-Q42*II-1	II-1 AOC13/R12 SS-R12*II-1	II-1 AOC13/R31 SS-R31*II-1	II-1 AOC13/S15 SS-S15*II-1	II-1 AOC13/S34 SS-S34*II-1
	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
	4/6/92	4/7/92	4/7/92	4/7/92	4/7/92	4/7/92	8/4/93	4/6/92	4/6/92	4/7/92	4/6/92	4/6/92	4/6/92	4/7/92	4/6/92	4/7/92
	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q
PCB-1016	R	R	R	R	R	0.37 U	14 U	R	0.034 U	R	0.068 U	0.036 U	0.035 U	0.034 U	0.035 U	R
PCB-1221	R	R	R	R	R	0.74 U	24 U	R	0.069 U	R	0.14 U	0.073 U	0.072 U	0.068 U	0.072 U	R
PCB-1232	R	R	R	R	R	0.37 U	14 U	R	0.034 U	R	0.068 U	0.036 U	0.035 U	0.034 U	0.035 U	R
PCB-1242	R	R	R	R	R	0.37 U	14 U	R	0.034 U	R	0.068 U	0.036 U	0.035 U	0.034 U	0.035 U	R
PCB-1248	28 J	5.6 J	5.9 J	3.4 J	0.02 J	6.1	150	430 J	0.034 U	R	0.068 U	0.085	0.035 U	0.034 U	0.035 U	4.4 J
PCB-1254	61 J	31 J	26 J	25 J	0.31 J	11	14 U	R	0.46	35 J	0.78	0.16	0.86	0.21	1.1	35 J
PCB-1260	R	R	R	R	R	0.37 U	14 U	R	0.034 U	R	0.068 U	0.036 U	0.035 U	0.034 U	0.035 U	R
	II-1 AOC13/T20 SS-T20*II-1	II-1 AOC13/U17 SS-U17*II-1	II-1 AOC13/U28 SS-U28*II-1	II-1 AOC13/U36 SS-U36*II-1	II-1 AOC13/V23 SS-V23*II-1	II-1 AOC13/W13 SS-DUP3*II-1	II-1 AOC13/W13 SS-W13*II-1	II-1 AOC13/W32 SS-W32*II-1	II-1 AOC13/Y15 SS-Y15*II-1	II-1 AOC13/Y21 SS-Y21*II-1	II-1 AOC13/Z28 SS-Z28*II-1					
	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5					
	1	1	1	1	1	1	1	1	1	1	1					
	4/6/92	4/6/92	4/6/92	4/6/92	4/6/92	4/6/92	4/6/92	4/7/92	4/6/92	4/6/92	4/6/92					
	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q	Result	Q
PCB-1016	R	0.036 U	0.35 U	R	0.036 U	0.035 U	0.034 U	0.042 U	0.36 U	0.36 U	0.18 U					
PCB-1221	R	0.072 U	0.71 U	R	0.074 U	0.07 U	0.069 U	0.085 U	0.74 U	0.73 U	0.37 U					
PCB-1232	R	0.036 U	0.35 U	R	0.036 U	0.035 U	0.034 U	0.042 U	0.36 U	0.36 U	0.18 U					
PCB-1242	R	0.036 U	0.35 U	R	0.036 U	0.035 U	0.034 U	0.042 U	0.36 U	0.36 U	0.18 U					
PCB-1248	R	0.036 U	0.35 U	4.1 J	0.036 U	0.035 U	0.034 U	0.042 U	0.36 U	0.65	0.18 U					
PCB-1254	58 J	0.036 U	5.6	31 J	0.4	0.043 J	0.068 J	0.055	7.6	6.8	1.8					
PCB-1260	R	0.036 U	0.35 U	R	0.036 U	0.035 U	0.034 U	0.042 U	0.36 U	0.36 U	0.18 U					

All results reported in mg/kg (ppm).
All nondetected results reported at full detection limits.
U - undetected.
J - Estimated result.
D - Diluted sample
R - Rejected result.

TABLE 3-4
WARWICK AREA
SWMU-5
PCBs IN SURFACE AND BORING SAMPLES

1/31/95

PHASE/ROUND SUR AREA / LOCATION SAMPLE ID	IR-2 SMU5/Y3 SF-S5-Y3(S)*IR-2	II-1 SMU5/R5A1 B-5A1*II-1	II-1 SMU5/R5B1 R-5B1*II-1	II-1 SMU5/R5C1 R-5C1*II-1	II-1 SMU5/R5D1 R-5D1*II-1	II-1 SMU5/R5E1 R-5E1*II-1	II-1 SMU5/R5F1 B-5F1*II-1	II-2 SMU-5/SG1 B-5G1*II-2	II-2 SMU-5/SG1 B-DUP1*II-2	II-2 SMU-5/SH1 B-5H1*II-2
DEPTH FROM (FT)	0.5	0	0	0	0	0	0	0	0	0
DEPTH TO (FT)	1	2	2	2	2	2	2	2	2	2
COLLECT DATE	3/19/91	7/28/93	7/28/93	7/28/93	7/28/93	7/28/93	7/28/93	7/29/93	5/5/94	5/5/94
	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q
PCB-1016	0.012 U	0.17 U	0.071 U	1.9 U	0.35 U	0.07 U	0.035 U	0.18 U	0.39 U	0.078 U
PCB-1221	0.023 U	0.35 U	0.14 U	3.4 U	0.71 U	0.14 U	0.072 U	0.38 U	0.79 U	0.16 U
PCB-1232	0.023 U	0.17 U	0.071 U	1.9 U	0.35 U	0.07 U	0.035 U	0.18 U	0.39 U	0.078 U
PCB-1242	0.012 U	0.17 U	0.071 U	1.9 U	0.35 U	0.07 U	0.035 U	0.18 U	0.39 U	0.078 U
PCB-1248	0.012 U	0.17 U	0.071 U	1.9 U	8.1 J	0.07 U	0.035 U	0.18 U	0.39 U	0.078 U
PCB-1254	0.71	0.72	0.28 J	4.9 J	6.1 J	0.07 U	0.035 U	0.18 U	0.39 U	0.21
PCB-1260	0.023 U	0.17 U	0.071 U	1.9 U	0.35 U	0.07 U	0.035 U	0.18 U	0.39 U	0.078 U
	IR-1 SMU5/C1 SF-S5-C1(D)*IR-1	IR-1 SMU5/C2 SF-S5-C2(D)*IR-1	IR-1 SMU5/C3 SF-S5-C3(D)*IR-1	IR-1 SMU5/D2 SF-S5-D2(D)*IR-1	IR-1 SMU5/D3 SF-S5-D3(D)*IR-1	IR-2 SMU5/A2 SF-S5-A2(D)*IR-2	IR-2 SMU5/B3 SF-S5-B3(D)*IR-2	IR-2 SMU5/C1 SF-S5-C1(D)*IR-2	IR-2 SMU5/C2 SF-S5-C2(D)*IR-2	IR-2 SMU5/C4 SF-S5-C4(D)*IR-2
	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5
	2	2	2	2	2	2	2	2	2	2
	11/15/90	11/15/90	11/15/90	11/15/90	11/15/90	3/19/91	3/19/91	3/19/91	3/19/91	3/19/91
	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q
PCB-1016	R	25 U	0.61 U	1.2 U	2.6 U	0.012 U	0.58 U	1.2 U	R	0.011 U
PCB-1221	R	50 U	1.2 U	2.5 U	5.1 U	0.023 U	1.2 U	2.3 U	R	0.021 U
PCB-1232	R	50 U	1.2 U	2.5 U	5.1 U	0.023 U	1.2 U	2.3 U	R	0.021 U
PCB-1242	R	25 U	0.61 U	1.2 U	2.6 U	0.012 U	0.58 U	1.2 U	R	0.011 U
PCB-1248	R	25 U	0.61 U	1.2 U	2.6 U	0.012 U	0.58 U	1.2 U	49	0.011 U
PCB-1254	R	50 U	1.2 U	2.5 U	5.1 U	0.023 U	12	36 J	R	0.073
PCB-1260	R	50 U	1.2 U	2.5 U	5.1 U	0.023 U	1.2 U	2.3 U	R	0.021 U
	IR-2 SMU5/E3 SF-S5-E3(D)*IR-2	IR-2 SMU5/Z3 SF-S5-Z3(D)*IR-2	II-1 SMU5/B5A2 B-5A2*II-1	II-1 SMU5/B5R2 B-5R2*II-1	II-1 SMU5/B5C2 B-5C2*II-1	II-1 SMU5/B5D2 B-5D2*II-1	II-1 SMU5/B5E2 B-5E2*II-1	II-1 SMU5/B5F2 B-5F2*II-1	II-2 SMU-5/SG2 B-5G2*II-2	II-2 SMU-5/SH2 B-5H2*II-2
	1.5	1.5	2	2	2	2	2	2	2	2
	2	2	4	4	4	4	4	4	4	4
	3/19/91	3/19/91	7/28/93	7/28/93	7/28/93	7/28/93	7/28/93	7/29/93	5/5/94	5/5/94
	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q	Result Q
PCB-1016	0.063 U	R	0.038 U	0.7 U	4.4 U	0.037 U	0.034 U	0.036 U	8.1 U	0.037 U
PCB-1221	0.13 U	R	0.077 U	1.4 U	8.9 U	0.075 U	0.07 U	0.073 U	16 U	0.075 U
PCB-1232	0.13 U	R	0.038 U	0.7 U	4.4 U	0.037 U	0.034 U	0.036 U	8.1 U	0.037 U
PCB-1242	0.063 U	R	0.038 U	0.7 U	4.4 U	0.037 U	0.034 U	0.036 U	8.1 U	0.037 U
PCB-1248	0.063 U	160 J	0.038 U	0.7 U	4.4 U	0.037 U	0.034 U	0.036 U	8.1 U	0.037 U
PCB-1254	0.13 U	R	0.14 J	0.7 U	4.4 U	0.037 U	0.034 U	0.036 U	8.1 U	0.037 U
PCB-1260	0.13 U	R	0.038 U	0.7 U	4.4 U	0.037 U	0.034 U	0.036 U	8.1 U	0.037 U

All results reported in mg/kg (ppm).
All nondetected results reported at full detection limits.
U - undetected.
J - Estimated result.
D - Diluted sample.
R - Rejected result.

**TABLE 3-5
WARWICK AREA
SWMU-6
ZINC IN SOIL SAMPLES**

PHASE / ROUND	IB-1	IB-2	IB-2	IB-2
	SUB AREA SMU6/Y5	SMU6/A1	SMU6/B1	SMU6/Y5
	SAMPLE ID SF-S6*IB-1	SF-S6-A1*IB-2	SF-S6-B1*IB-2	SF-S6*IB-2
	COLLECT DATE 11/14/90	3/12/91	3/12/91	3/12/91
ZINC	Result Q	Result Q	Result Q	Result Q
	850 J	56.7 J	111 J	2390 J

BLDG 15

MILL
ST.

BRIDGE

PAWTUXET RIVER

- ⊕ Boring (A)-PROD
- Surface Soil-PROD

ft (1 in = 70 ft)

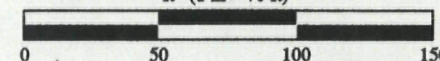
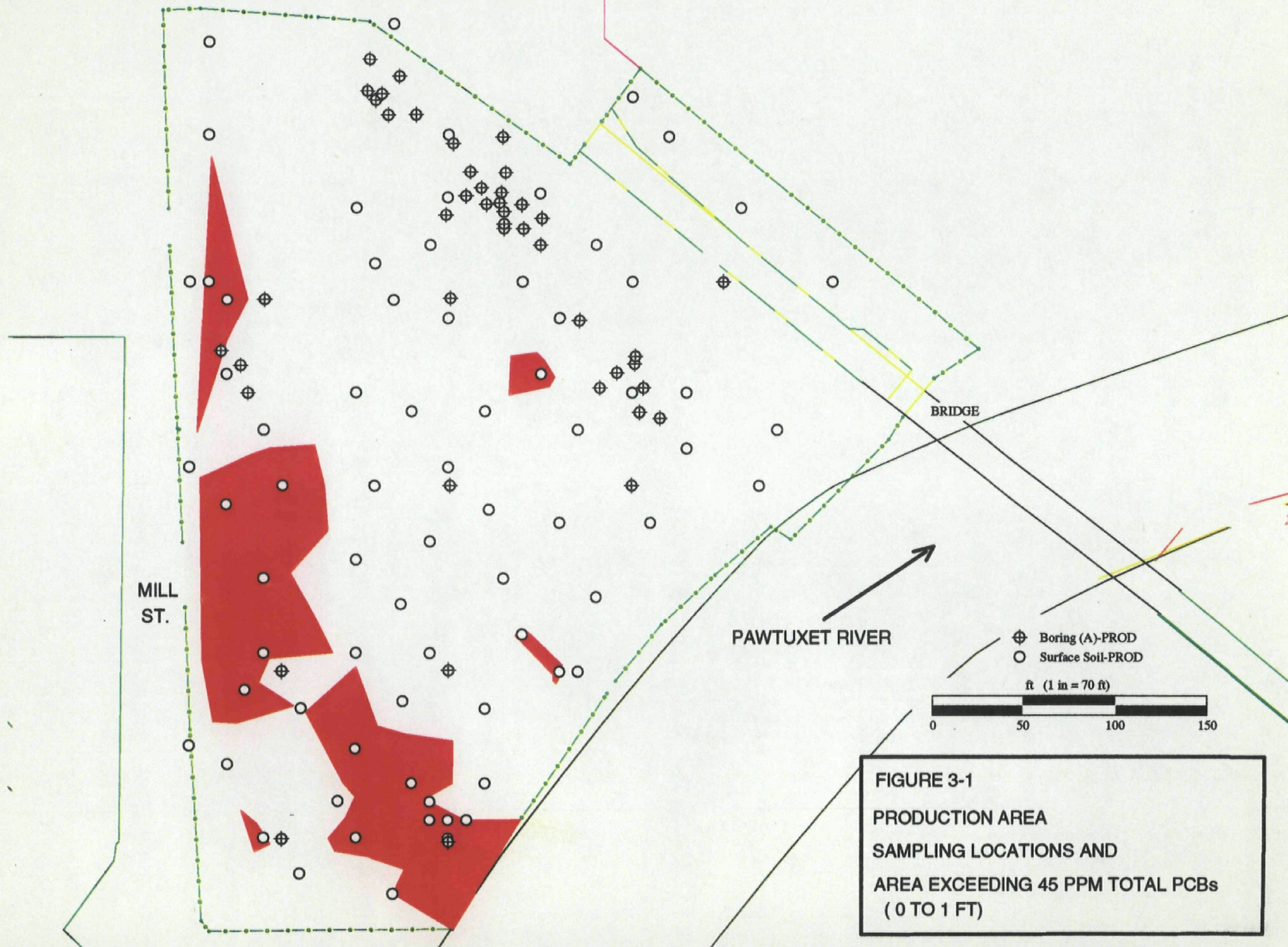
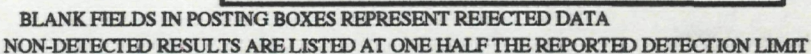


FIGURE 3-1
PRODUCTION AREA
SAMPLING LOCATIONS AND
AREA EXCEEDING 45 PPM TOTAL PCBs
(0 TO 1 FT)



IRM CLEANUP LEVEL (1 PPM) IS FOR TOTAL PCBs



PRELIMINARY TASKS

4.1 OVERVIEW

This chapter describes the preliminary tasks which will be performed as part of these IRMs. These tasks include identifying the limits of areas to be excavated, identifying permit requirements, and classifying the soil for waste characteristics.

Section 4.2 discusses identifying areas to be excavated. Section 4.3 discusses identifying permit requirements. Section 4.4 discusses classifying the soil for waste characteristics.

4.2 IDENTIFYING AREAS OF EXCAVATION

Excavations will be performed in three areas: 1) the Production Area; 2) SWMU-5; and, 3) SWMU-6. The limits of the excavations for the Production Area and SWMU-5 were determined by evaluating analytical data for soil sampled during the RFI. The limits of excavation for SWMU-6 will be determined in the field at the time of removal. The zinc oxide/soil pile (SWMU-6) has been formed into an easily identifiable berm.

4.2.1 Production Area

Figure 3-1 shows the limits of the Production Area and the location of the surface soil samples and soil borings. Figure 4-1 shows the locations of the former buildings in the Production Area. Three types of foundations were used in this area: 1) slab on grade supported by pilings; 2) poured concrete foundations with one basement level supported by pilings; and, 3) floating concrete foundations several feet thick. Underground utilities (e.g., electrical conduit, sanitary sewer, and water lines) are reportedly about 4 ft below ground surface. There is a large concentration of buried lines in the main north-south corridor through the Production Area. Reportedly, the areas between the buildings were almost entirely asphalt paved.

The Production Area was gridded as part of the initial investigation. Selected grid nodes will be re-established in the proposed excavation areas prior to the start of the field effort. Control points will be established beyond the working area to facilitate re-establishing the grid as necessary during the course of the excavation.

The limits of the areas exceeding 45 ppm total PCBs (the IRM cleanup level for the Production Area) are shown on Figure 3-1. The proposed area of excavation shown on Figure 4-1 was established by "squaring off" the 45 ppm isoconcentration boundary to create a limit of excavation that is more representative of the finite limits of

working with heavy equipment.

The final limits of the excavation will be established based on the grid system. The limits of the excavation will not be surveyed. Only the locations of post-excavation samples will be horizontally located by a surveyor to +/- one foot.

4.2.2 SWMU-5

Figure 3-2 shows the locations of the soil samples from SWMU-5. The sample results from these locations were contoured, as described in Section 3.3, to establish the limits of the area which included concentrations greater than 1 ppm, the proposed IRM cleanup level for PCBs at SWMU-5.

The limits of the area estimated to exceed 1 ppm PCBs also are shown on Figure 3-2. The proposed area of excavation shown on this figure was established by visually delineating the area where actual PCB concentrations exceed 1 ppm (discussed in Section 3.3).

Horizontal survey control points will be established adjacent to the excavation area. These control points will be used to establish the perimeter shown on Figure 3-2. The limits of the excavation will not be surveyed. Only the locations of post-excavation samples will be horizontally located by a surveyor to +/- one foot.

4.3 PERMITTING

The initial task in the implementation of these IRMs will be the identification of federal, state and local permits required for the implementation of the soil excavation. A preliminary review of federal, state and local permit requirements for the PCB driven excavations include, but may not be limited to:

Federal

1. TSCA-PCBs

Spills of PCBs in excess of 50 ppm are subject to TSCA regulations. PCB contaminated wastes will have to be included on the uniform hazardous waste manifest. Ciba will need to notify USEPA of PCB waste activity if they own or operate a storage facility for PCBs designated for disposal.

2. RCRA - Land Disposal Restrictions

If any of the soils fail TCLP, they may require treatment prior to disposal.

State Permits

1. Air Pollution Control Monitoring Requirements/Permits

RIDEM will be contacted to determine if air pollution monitoring, beyond that proposed in this Work Plan, will be required for the excavation activities.

2. Spill Prevention

The assumption is that spill prevention requirements are not applicable to small excavation activities. These requirements will be reviewed with the appropriate state agency.

3. Freshwater Wetlands Permits

Activities within the Production Area and SWMU-5 are subject to the RI Freshwater Wetlands Law. Activities within the 100 year floodplain and/or within 200 feet of the bank of a river which is greater than 10 feet in width on average require a permit. Riverbank is defined in the regulations as "that area of land within 200 feet of the edge of any flowing body of water having a width of 10 feet or more." Permits and/or exemptions will have to be obtained from RIDEM prior to the initiation of field activities.

Local Permits

The following is a list of other potential local regulatory issues which may impact these IRMs:

1. Site plan approval
2. Local zoning requirements
3. Soil erosion and sedimentation control
4. Local requirements for activity in the 100 year floodplain
5. General construction permits

4.4 WASTE CLASSIFICATION

Waste streams from three areas will be generated: 1) Production Area; 2) SWMU-5; and, SWMU-6. The subsequent sections will present a preliminary review of data and assumptions critical to proper classification of these waste streams.

4.4.1 Production Area

The suites of compounds of concern for this area are volatile organic compounds (VOCs), pesticides and PCBs. Our current understanding of the sources of these compounds are as follows:

- VOCs were used in the intermediate steps of batch production processes. The VOCs came into contact with soil and groundwater through minor discharges within the plant buildings in the normal course of operations (e.g., small leaks in seals or small spills during transfer or evacuation of process equipment. These de minimis releases may have been flushed into floor drains and/or sumps during normal plant maintenance operations.
- PCBs were derived from two sources; 1) de minimis releases of Dowtherm A, a heat transfer fluid, within process buildings during production; and, 2) de minimis releases of PCB containing hydraulic fluid within process buildings during production. These de minimis releases may have been flushed into floor drains and/or sumps during normal plant maintenance operations.
- Pesticides were applied to plant areas during normal maintenance operations at prescribed concentrations. Pesticides do not reflect releases of intermediate or final products of plant processes. The pesticide compounds found in the soil were not produced at the Site.

On the basis of the historical information summarized above, the analytical results from characterization/delineation sampling, and the preliminary waste classification sampling results, the excavated material from the Production Area will not be classified as a hazardous waste unless TCLP or RCRA characteristic limits are exceeded.

4.4.2 SWMU-5

The suites of compounds of concern for this area are VOCs, pesticides and PCBs. Our current understanding of the sources of these compounds are as follows:

- VOCs were derived from de minimis quantity releases (Section 4.4.1) from the Production Area which were flushed into floor drains and/or sumps which ultimately discharged to the former Coffey Dam area in the Pawtuxet River. Impacted sediment generated from the removal of the Coffey Dam was staged at SWMU-5. These sediments were removed in 1976 but there was a residual impact to underlying soils which was not addressed at the time the sediment was removed from

SWMU-5.

- PCBs were derived from de minimis quantity releases (Section 4.4.1) from the Production Area which were flushed into floor drains and/or sumps which ultimately discharged to the former Coffey Dam area in the Pawtuxet River. Impacted sediment generated from the removal of the Coffey Dam was staged at SWMU-5. These sediments were removed but there was a residual impact to underlying soils which was not addressed at the time the sediment was removed from SWMU-5.
- Pesticides may have been applied to plant areas during normal maintenance operations at prescribed concentrations. Pesticides do not reflect releases of intermediate or final products of plant processes. The pesticide compounds found in the soil were not produced at the Site.

On the basis of the historical information summarized above, the analytical results from characterization/delineation sampling, and the preliminary waste classification sampling results, the excavated soil from SWMU-5 area will not be classified as a hazardous waste unless TCLP or RCRA characteristic limits are exceeded.

4.4.3 SWMU-6

On the basis of historical information about SWMU-6 and on analytical results from characterization/delineation sampling, the excavated soil from this area will be classified as a non-hazardous waste.

4.4.4 Potential Impact of the Universal Treatment Standards (UTS)

Waste disposal also may be impacted by the September 14, 1994 Land Disposal Restriction regulations. This rule promulgates treatment standards for newly identified organic toxicity characteristic wastes D018-D043. This rule may potentially impact the disposal of soil from within these areas if the soil fails the final TCLP analysis. An exceedance of a TCLP criteria could mean the waste would have to be treated before it is landfilled to reduce the concentration of the selected contaminant(s) to the Universal Treatment Standards. These standards would be applied both to the compound(s) that failed TCLP as well as all underlying hazardous constituents (UHCs).

For the purpose of this Work Plan, the assumption has been made that all waste will pass TCLP (as indicated by the results of the preliminary waste classification sampling results). No treatment will be necessary or performed at the Site. All soil will be disposed of in accordance with federal, state, and local regulations.

4.5 IN-SITU WASTE CLASSIFICATION

Waste classification samples will be collected and analyzed before the start of soil excavation activities. Proposed waste classification composite locations are shown on Figures 4-2 and 4-3. Individual sample collection points for these composites will be biased towards areas of elevated PCB concentrations. These proposed sampling locations may change based on conditions encountered in the field. The sampling scheme will be as follows:

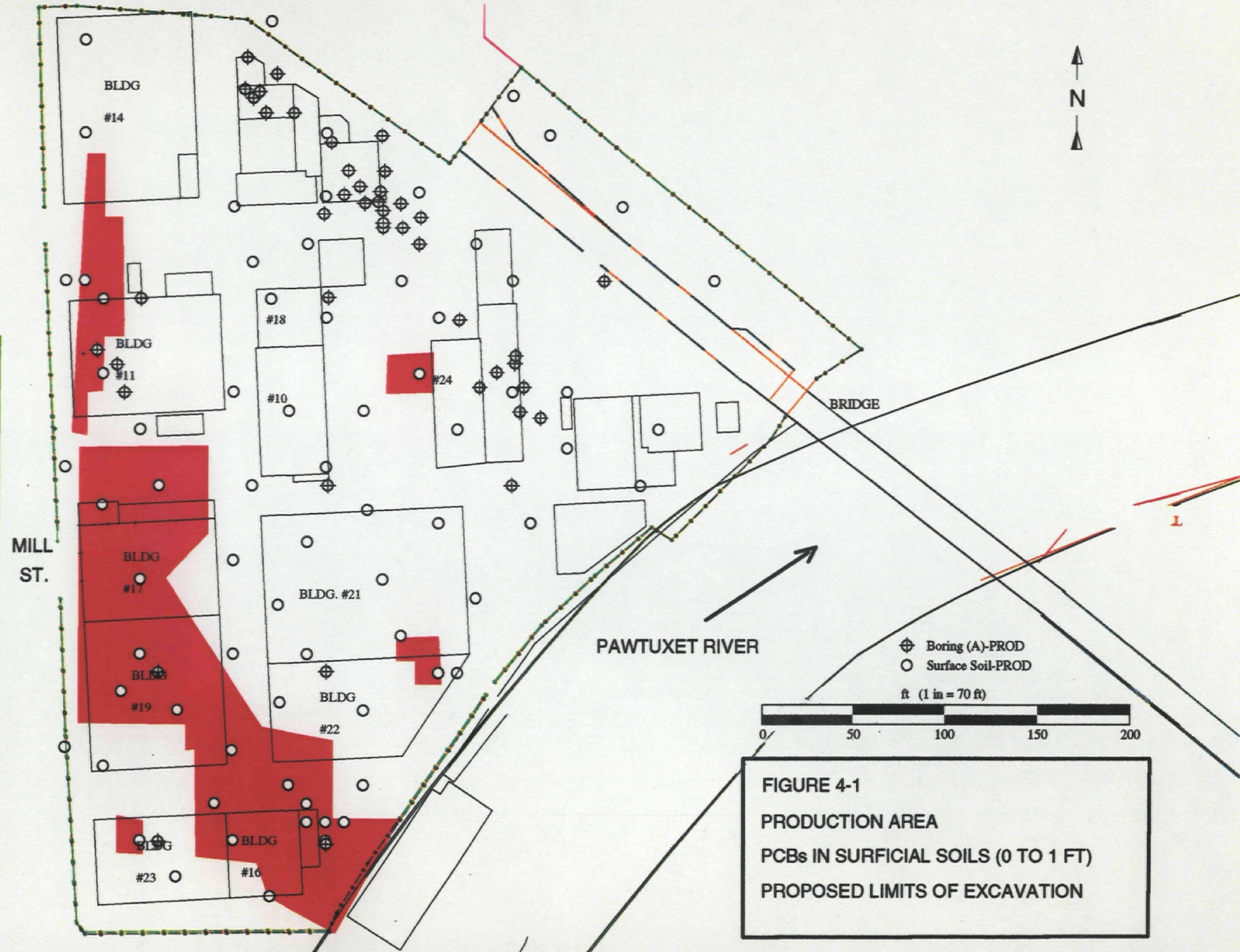
- one sample per 20 cubic yards will be composited into a single sample representative of not more than 100 cubic yards;
- samples from the Production Area will be collected from 0.5-1.0 ft below ground surface because the analytical results suggest the contamination is concentrated within this horizon;
- samples from SWMU-5 will be collected from the 0-2 ft horizon;
- one sample will be collected for SWMU-6; and
- all samples will be analyzed for RCRA Characteristics, TCLP, and total PCBs.

If a composite sample fails the RCRA Characteristics or TCLP analyses, the individual samples (used to make up the composite) may be reanalyzed to evaluate the specific source of contamination. Also, additional samples may be collected to isolate "hot spots" if encountered.

The results of the TCLP analyses combined with the descriptions of the waste streams presented in the preceding sections will be used to generate an appropriate waste classification for review by potential waste disposal facilities. In addition, it may be necessary to obtain additional soil for analyses specific to the requirements of the selected disposal facility.

BLDG 15

VOLUME OF SOIL EXCEEDING 45 PPM TOTAL PCBs ~ 779 CUBIC YARDS (BASED ON A 1 FT DETPH)



BLDG 15

MILL
ST.

BRIDGE

PAWTUXET RIVER

ft (1 in = 70 ft)

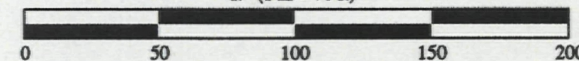


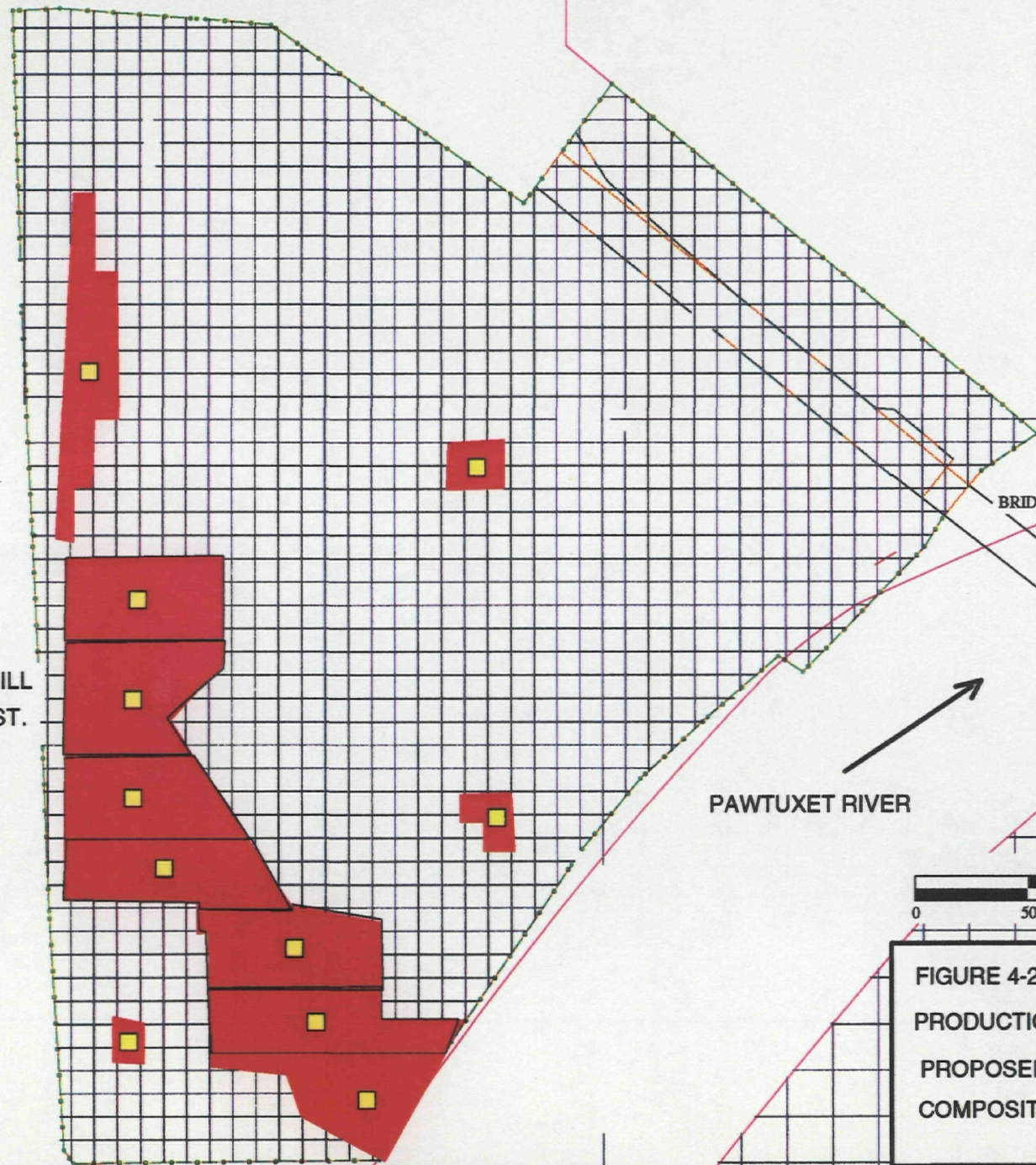
FIGURE 4-2

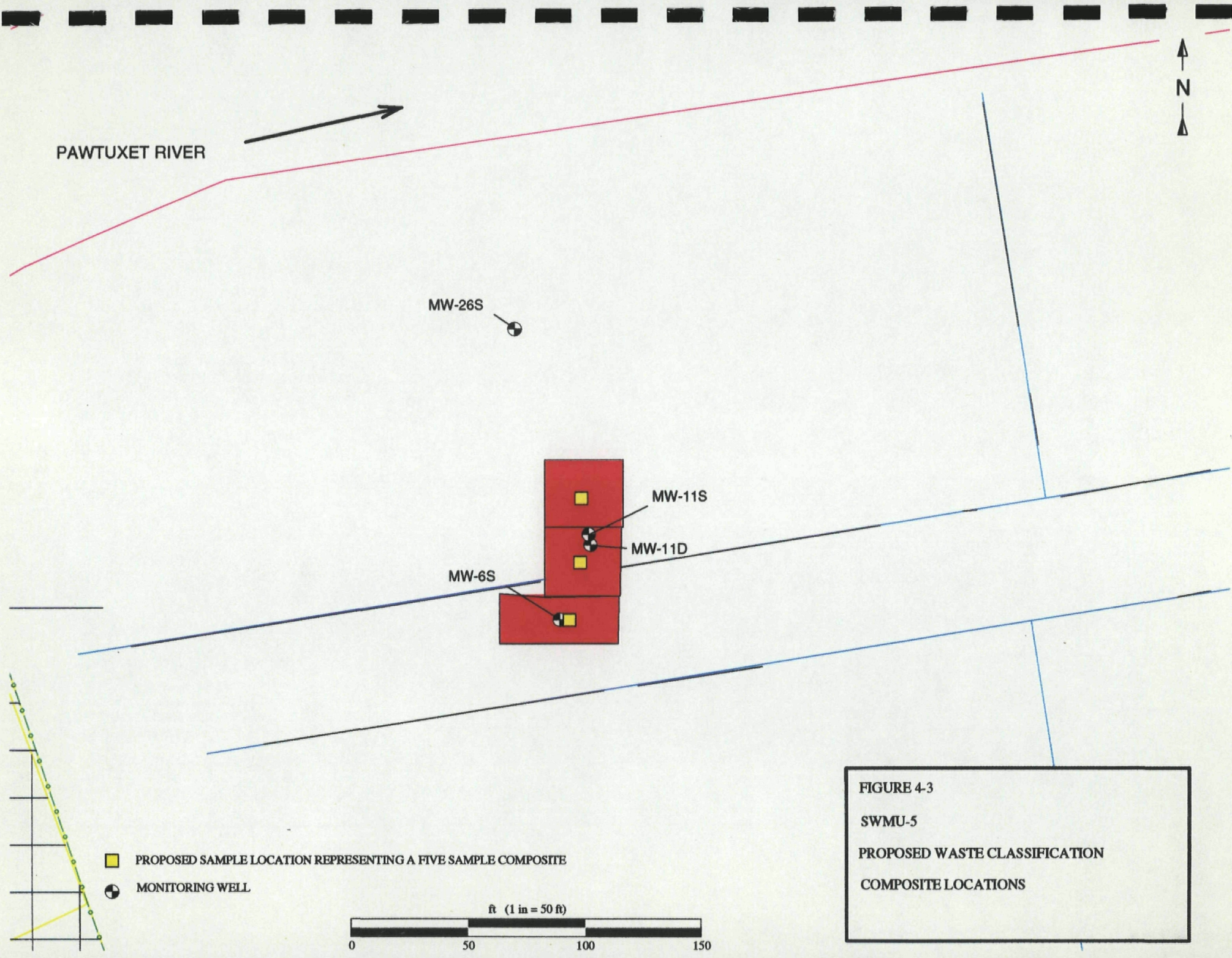
PRODUCTION AREA

PROPOSED WASTE CLASSIFICATION

COMPOSITE LOCATIONS

■ PROPOSED SAMPLE LOCATION REPRESENTING FIVE COMPOSITE SAMPLES





5.1 OVERVIEW

This chapter presents the scope of work for the IRM that will be implemented in the Production Area. Existing analytical data for soil samples collected in the Production Area were presented in Section 3.2. For this IRM, excavation activities will be limited to soils with PCB concentrations above the IRM cleanup level (45 ppm). The lateral limits of areas exceeding 45 ppm PCBs were described in Section 4.2 and shown on Figure 3-1. These areas will be excavated to a depth of about one foot below ground surface.

Section 5.2 discusses the approach and assumptions for this IRM being implemented in the Production Area. Section 5.3 describes excavation activities. Section 5.4 discusses disposal of the excavated soil.

5.2 APPROACH AND ASSUMPTIONS

The approach for implementing this IRM for soils in the Production Area was designed using the following assumptions:

- The depth of the initial excavation generally will be limited to about one-foot. If necessary, additional excavation will be performed in areas that exceed the IRM cleanup level (based on post-excavation sampling results). Additional excavation, if required, generally will not exceed 2 feet below ground surface;
- No excavations will be performed below the foundations of former buildings, including both slab on grade and floating slab construction. In addition, construction debris, exclusive of the first foot of debris, will not be removed from within the foundations of former basements (i.e. construction rubble generated when buildings were collapsed within the former basements);
- No remaining building foundations or floor slabs will be removed. These structures will be left in place and will be addressed (if necessary) in the development of a final remedy for this area;
- No concrete, building stone, piping or other construction debris will be removed from the Production Area if the longest dimension is greater than approximately 1 ft on any axis (visual estimate). Any such material

will be left on-site at the discretion of Ciba or its representative;

- Mechanical (mechanized) materials sorting will not be used on any excavated material (due to dust generation and health and safety concerns);
- All excavated material will be disposed at a licensed RCRA/TSCA landfill (hazardous or non-hazardous, depending on the results of the TCLP analyses);
- A qualified remedial contractor will conduct the excavation activities with oversight by Ciba or its oversight contractor;
- All field activities will be performed by qualified personnel with 40 hour OSHA training, 8 hour refreshers (if necessary) and participation in a medical monitoring program. All work will be performed in modified Level D personal protection equipment (as required in the IRM addendum to the existing Health and Safety Plan);
- Fugitive dust will be minimized using simple misting/watering devices (e.g. a mister on a hose from a potable water supply); and
- Fugitive dust monitoring will be limited to periodic monitoring using manual equipment specified in the Health and Safety Plan. Continuous monitoring will not be performed for this IRM.

5.3 EXCAVATION ACTIVITIES

Before excavating any soils in the Production Area, soil samples will be collected for waste classification as described in Section 4.5. Excavation activities will be conducted by a qualified remedial contractor to be selected by Ciba with oversight by Ciba or its authorized representative.

5.3.1 Excavation

The proposed limits of areas to be excavated in the Production Area are shown on Figure 4-1. These areas will be excavated using conventional methods (e.g., backhoe) to an initial depth of about one foot. If physical barriers are encountered, Ciba reserves the right to implement alternative excavation methods (e.g., vacuuming, sweeping). Physical barriers or other limiting factors that may impact excavation activities within the Production Area may include, but are not limited to the following:

- "Grade beams"- concrete slabs were reported to be anchored to grade beams which formed horizontal grids beneath the floor slabs to provide support. The spacing of these beams is not known. If encountered, these grade beams will not be removed. Excavation will take place within the area defined by these grade beams;
- "Pile caps"- grade beams were supported on pile caps. These caps are reported to be 3-4 feet below grade; pile caps should not impact excavation activities. If encountered, the pile caps will be left in place and will be treated as obstructions;
- "Strengthened slabs"- thicker concrete slabs containing additional support members incorporated into floor slabs. These sections were used typically to support heavy equipment (e.g., boilers, generators, etc.). The strengthened slab sections will not be removed and will be treated as obstructions;
- "Pipe conduits"- or pipe tunnels are primary paths for sewer, water, and process underground pipes. It is understood that these conduits are at least three feet below grade and should not impact excavation activities. If encountered, the pipe conduits will be treated as obstructions and left in place;
- "Bulkhead"- the bulkhead adjacent to the Production Area will not be disturbed. Excavation will be stopped if on-site inspection determines the stability or integrity of the bulkhead may be compromised;
- "Foundation walls"- from buildings with basements are expected to be encountered. These will be left in place and treated as obstructions; and
- "City of Cranston right-of-way"- the proposed limits of excavation do not currently abut the right-of-way. The excavation will not be extended into the right-of-way unless permission is obtained from the City.

Because excavation activities in this area are expected to proceed slowly, soils may be stockpiled temporarily (generally not to exceed 48 hours) in the Production Area. Any stockpiled soils will be covered with reinforced poly-sheeting. Soils will be loaded into lined dump trailers or roll-off containers for transportation to the disposal facility. Full containers will be secured with reinforced polyethylene tarps (or equivalent) and temporarily staged within a secure area. Trailers or roll-off containers will be transported off site in a timely manner. All stored and transported material will be properly labeled for PCBs.

5.3.2 Post-Excavation Sampling

Post-excavation sampling will be in accordance with the document "Field Manual for Grid Sampling of PCB Spill Sites to Verify Cleanup" (EPA-560/5-86-017). As per this document, because the excavations in the Production Area and SWMU-5 will be greater the 400 square feet in area, a maximum of 37 confirmatory post-excavation samples will be collected in each area. The sampling locations will be distributed in a hexagonal grid pattern (Figure 5-1). Compliance with the cleanup standard will be considered achieved when the following criteria are met:

- the 95th upper confidence limit of the mean of the sample concentrations is less than or equal to the standard;
- no single sample exceeds the standard by a factor of ten; and
- no more than ten percent of the individual samples exceed the standard.

Soil samples will be scraped from the top inch of the exposed soil at each sampling location. The sampling area (not depth) will be extended as necessary to obtain adequate sample volume. A stainless steel trowel will be used to collect the sample. Samples will be transferred directly from the trowel to a laboratory container. The container will be labeled and stored in an ice chest at 4 degrees Centigrade.

If a sampling location coincides with a concrete obstruction in the excavation, the concrete will be sampled by coring the top inch of the surface of the concrete. Adjacent one-inch cores will be collected until adequate sample volume is obtained. The cores will be transferred directly into a laboratory container. The container will be labeled and stored in an ice chest at 4 degrees Centigrade.

If a sampling location coincides with a non-porous obstruction in the excavation, a wipe sample will be collected. Wipe samples will be collected by wiping a 100-cm² area with a solvent soaked gauze pad or 11 cm filter paper. The area to be wiped will be marked with masking tape. Disposable rubber gloves will be used to hold the gauze pad during wiping. The gauze pad will be placed in the sample jar supplied by the laboratory, labeled and stored in an ice chest at 4 degrees Centigrade.

All samples will be labeled as PCB-containing material (yellow TSCA labels). Rubber gloves used for sampling will be discarded in plastic bags intended for PCB-contaminated materials.

Two field duplicates (one per 20 samples) will be collected for analysis to assess quality control.

Soil samples will be submitted to Savannah Laboratories, Inc., or Ciba's Environmental Testing Laboratory (use of ETL contingent upon USEPA approval). Each sample will be analyzed for PCBs by gas chromatography using EPA Method 8080 from SW-846. Concrete samples will be crushed by the laboratory prior to analysis to ensure proper solvent contact.

These samples will be collected and analyzed in accordance with the Quality Assurance Documents: Supplement (submitted in January 1992 and subsequently approved by USEPA).

5.3.3 Backfilling

Excavated areas in the Production Area will not be backfilled as part of this IRM unless backfilling is necessary to construct a parking lot on a portion of the Production Area. Final backfilling activities will be postponed until implementation of the final remedy.

5.4 DISPOSAL

All material will be disposed in accordance with applicable federal, state, and local regulations. All material will be disposed at a licensed RCRA/TSCA landfill (either as hazardous or non-hazardous based on results of waste classification sampling). Preliminary testing suggests that the waste will be classified as RCRA non-hazardous (see analytical results in Appendix C). Waste will be properly manifested and copies of the manifests will be submitted with the final IRM Report. Disposal facilities will be identified before excavation activities begin.

BLDG 15

MILL
ST.

BRIDGE

PAWTUXET RIVER

⊕ Point of Proposed Sampling Location
ft (1 in = 70 ft)

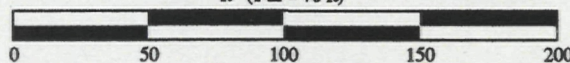
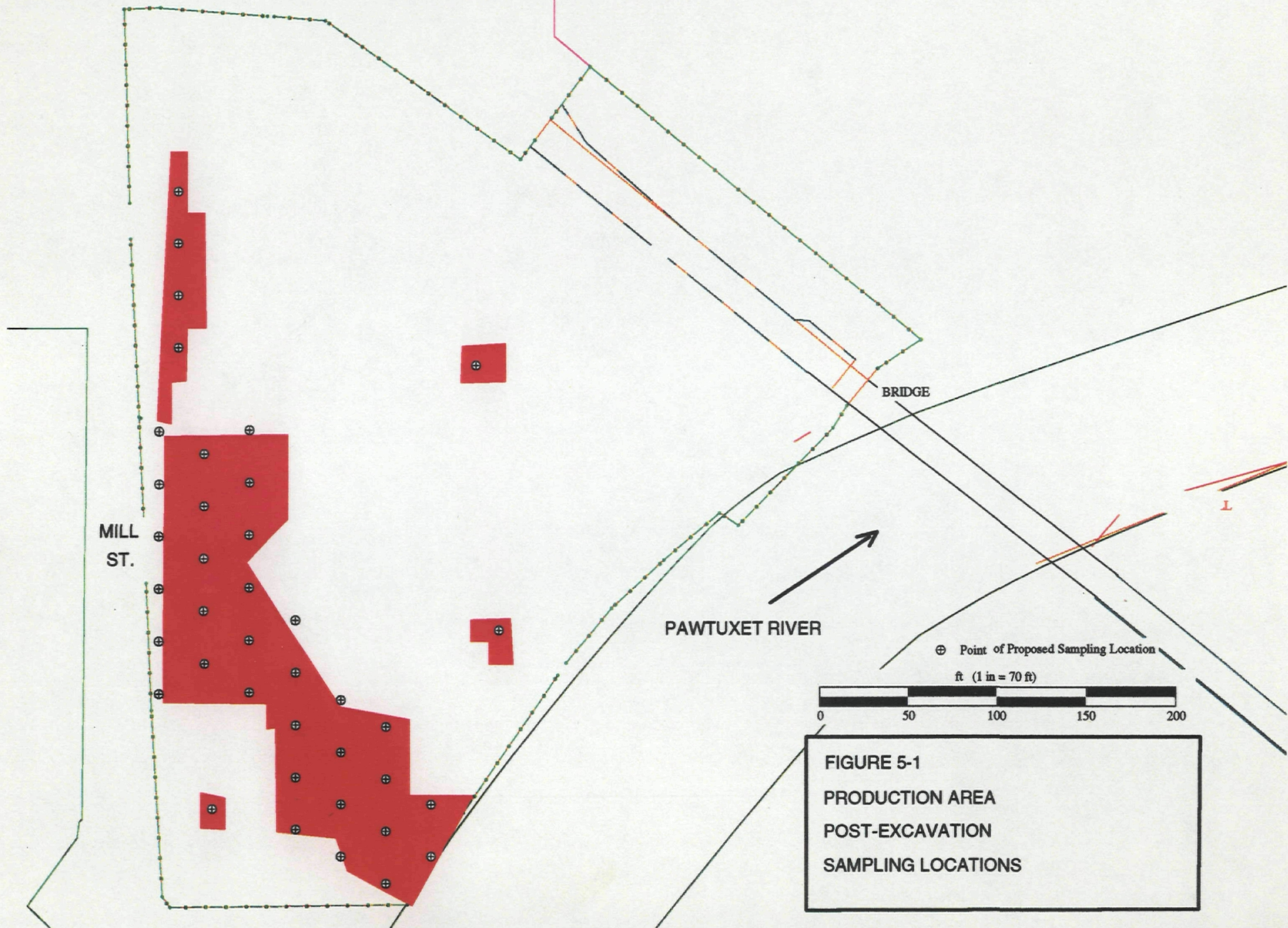


FIGURE 5-1
PRODUCTION AREA
POST-EXCAVATION
SAMPLING LOCATIONS



6.1 OVERVIEW

This chapter presents the scope of work for the Interim Remedial Measure (IRM) that will be implemented at SWMU-5 (located in the Warwick Area). Existing analytical data for soil samples collected in this area were presented in Section 3.3. For this IRM, excavation activities will be limited to soils with PCB concentrations above the EPA cleanup level for residential sites of 1 ppm. Areas of soil with PCB concentrations above the EPA cleanup level based solely on detection limits will not be excavated (see Section 3.3). The lateral limit of the area exceeding 1 ppm PCBs is shown on Figure 3-2. This area will be excavated to a depth of about two feet below ground surface.

Section 6.2 discusses the approach and assumptions for this IRM. Section 6.3 describes excavation activities to be performed. Section 6.4 discusses disposal of the excavated soil.

6.2 APPROACH AND ASSUMPTIONS

The approach for implementing this IRM at SWMU-5 was designed using the following assumptions:

- Waste classification sampling will be performed prior to the start of excavation. Samples will be collected a minimum of 60 days before the scheduled start of excavation;
- The depth of the excavation generally will be limited to two feet;
- Excavated areas will be backfilled with certified clean fill after the results of the post-excavation results have been reviewed;
- All excavated material will be disposed of at a licensed RCRA/TSCA landfill (hazardous or non-hazardous, depending on the results of the TCLP analyses);
- A qualified remedial contractor will perform the excavation activities with oversight by Ciba or Ciba's authorized representative;
- All field activities will be performed by qualified personnel with 40 hour OSHA training, 8-hour refreshers (if necessary) and participation in a medical monitoring program. All work will be performed in modified Level D personal protection equipment as specified in the

Health and Safety Plan Addendum;

- Fugitive dust monitoring will be limited to periodic monitoring using manual equipment specified in the Health and Safety Plan Addendum. Continuous monitoring will not be performed for this IRM.

6.3 EXCAVATION ACTIVITIES

Before excavating any soils in SWMU-5, soil samples will be collected for waste classification as described in Section 4.5. Excavation activities will be conducted by a qualified remedial contractor to be selected by Ciba with oversight by Ciba or its authorized representative.

6.3.1 Excavation

The proposed limits of the area to be excavated in SWMU-5 are shown on Figure 3-2. Vegetation will be cleared from this area prior to excavating. This area will be excavated to an initial depth of two feet using conventional methods (e.g., backhoe).

Ciba anticipates that the in-situ waste classification sampling results may show that all or portions of the area to be excavated are non-hazardous. Non-hazardous soils may be stockpiled in the concrete block foundation (former hazardous material storage area) located in the Warwick Area. Hazardous soils (if any) will be shipped off site immediately or stored in lined roll-offs covered with polyethylene tarps (or equivalent).

6.3.2 Post-Excavation Sampling

Post-excavation sampling will be in accordance with the document "Field Manual for Grid Sampling of PCB Spill Sites to Verify Cleanup" (EPA-560/5-86-017). As per this document, because the excavation in SWMU-5 will be greater the 400 square feet in area, a maximum of 37 confirmatory post-excavation samples will be collected in each area. The sampling locations will be distributed in a hexagonal grid pattern (Figure 6-1). Compliance with the cleanup standard will be considered achieved when the following criteria are met:

- the 95th upper confidence limit of the mean of the sample concentrations is less than or equal to the standard;
- no single sample exceeds the standard by a factor of ten;
- no more than ten percent of the individual samples exceed the standard.

Soil samples will be scraped from the top inch of the exposed soil at each sampling

location. The sampling area (not depth) will be extended as necessary to obtain adequate sample volume. A stainless steel trowel will be used to collect the sample. Samples will be transferred directly from the trowel to a laboratory container. The container will be labeled and stored in an ice chest at 4 degrees Centigrade.

All samples will be labeled as PCB-containing material (yellow TSCA labels). Rubber gloves used for sampling will be discarded in plastic bags intended for PCB-contaminated materials.

Two field duplicates (one per 20 samples) will be collected for analysis to assess quality control.

Soil samples will be submitted to Savannah Laboratories, Inc., or Ciba's Environmental Testing Laboratory (use of ETL contingent upon USEPA approval). Each sample will be analyzed for PCBs by gas chromatography using EPA Method 8080 from SW-846.

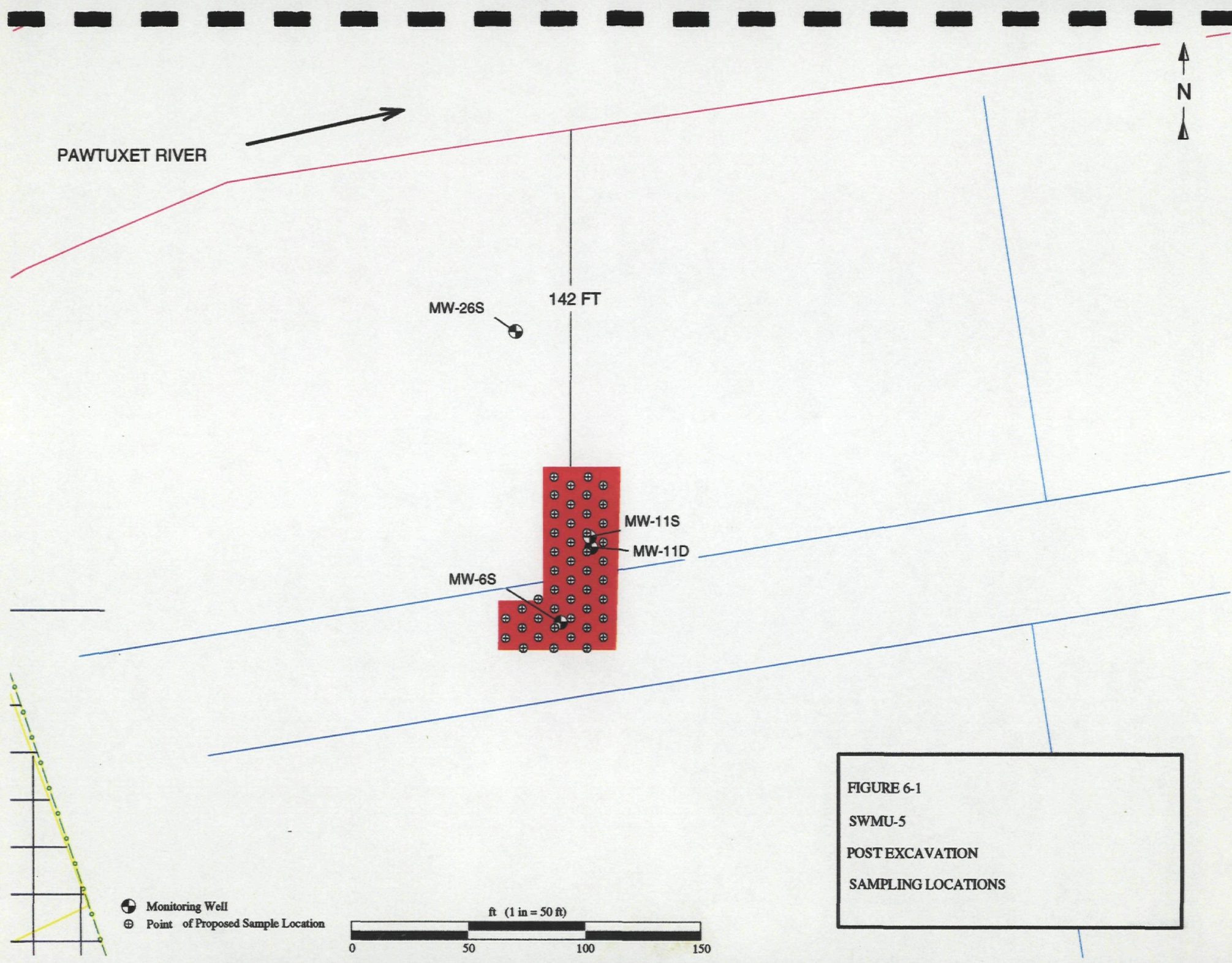
These samples will be collected and analyzed in accordance with the procedures in the Quality Assurance Documents (January 1992).

6.3.3 Backfilling

Excavated areas in SWMU-5 will be backfilled with certified clean fill after the confirmatory analytical results (from the laboratory) have been reviewed and evaluated.

6.4 DISPOSAL

All material will be disposed in accordance with applicable federal, state, and local regulations. All material will be disposed at a licensed RCRA/TSCA landfill (either as hazardous or non-hazardous based on results of waste classification sampling). Preliminary testing suggests that the waste will be classified as non-hazardous (see testing results in Appendix C). Waste will be properly manifested and copies of the manifests will be submitted with the final IRM Report. Disposal facilities will be identified before excavation activities begin.



WARWICK AREA SWMU-6 SOILS

7.1 OVERVIEW

This chapter presents the scope of work for the Interim Remedial Measure (IRM) that will be implemented at SWMU-6 (located in the Warwick Area). Existing analytical data for soil samples collected in this area were presented in Section 3.4. These data show zinc concentrations in surficial soils up to 2390 ppm. This area will be excavated using conventional methods (e.g., backhoe).

Section 7.2 discusses the approach and assumptions for this IRM. Section 7.3 describes excavation activities to be performed. Section 7.4 discusses disposal of the excavated soil.

7.2 APPROACH AND ASSUMPTIONS

The approach for implementing this IRM at SWMU-6 was designed using the following assumptions:

- Waste classification sampling will be performed prior to excavation. Samples will be collected a minimum of 60 days before the scheduled start of the load-out;
- The soil containing the zinc oxide will be loaded directly into lined dump trailers or roll-off containers for transportation and disposal;
- Approximately 6 inches of soil will be removed from beneath the stockpiles if the zinc oxide is in direct contact with the underlying soils. In areas in which the zinc oxide is on pavement, no additional material will be removed;
- No post-excavation samples will be collected;
- No fugitive dust monitoring will be performed;
- No backfill will be required;

7.3 EXCAVATION ACTIVITIES

Before excavating any soils at SWMU-6, one composite soil sample will be collected for confirmatory waste classification (as presented in Section 4.4). Excavation activities will be conducted by a qualified subcontractor to be selected by Ciba with

oversight by Ciba or its authorized representative.

7.3.1 Excavation

The proposed area to be excavated is defined by the physical shape of the soil berm. The location of SWMU-6 is shown on Figure 1-1. This berm is about 50-feet long by 7-feet wide by 2-feet high and contains about 25-30 cubic yards of soil.

All soil to be removed from SWMU-6 is anticipated to be non-hazardous. This material is currently stockpiled (bermed) on asphalt and partially on soil. The soil will be removed from the asphalt and the asphalt will be swept clean (i.e., no asphalt will be removed). In areas where the zinc oxide/soil pile is overlying soil, a maximum of six inches (of the underlying soil) will be removed.

Excavated soils will be loaded directly into lined dump trailers or roll-off containers for transportation and disposal at a licensed disposal facility.

7.4 DISPOSAL

All soil will be disposed in accordance with applicable federal, state, and local regulations. All soil may be disposed at a non-hazardous landfill. Waste will be properly manifested and copies of the manifests will be submitted with the final IRM Report. Disposal facilities will be identified before excavation activities begin.

8.1 OVERVIEW

Project management ensures that all work necessary for conducting this IRM will be completed in a timely fashion. A project management plan for the RCRA Facility Investigation was presented in Volume 1 of the RFI Proposal. That plan described the organization of the project and identified the tasks to be accomplished (including deliverable reports) as well as a schedule for completing those tasks. The project management plan was updated in subsequent plans including, but not limited to, the Phase I Interim Report and Phase II Proposal, the Phase II Pawtuxet River Proposal, the Stabilization Work Plan, etc.

This section updates the project management plan as it pertains to the activities described in the previous sections including;

- the *project organization* for this IRM (Section 8.2);
- the *schedule* for conducting the IRM (Section 8.3); and,
- the *reporting requirements* for this IRM (Section 8.4).

A summary concludes this chapter (Section 8.5).

8.2 ORGANIZATION

The Ciba Project Coordinator is responsible for 1) coordinating interaction among all project participants, and 2) ensuring that the objectives of this IRM are met. The organizational structure for this IRM is shown in Figure 8-1. Please note that the project coordinator for Ciba is now Dr. Barry Berdahl.

8.3 SCHEDULE

This IRM is on a separate schedule from all other ongoing site activities. The schedule for conducting this IRM is presented in Figure 8-2; it shows an estimated duration of 4.5 months. The schedule makes the following assumptions:

- USEPA will require 50 days to review this Work Plan;
- USEPA will only comment on the Work Plan. Ciba understands that this document will not be approved by USEPA;
- significant comments (if any) generated by USEPA during their review will be addressed in a single revised Work Plan or addenda;
- during USEPA's review, Ciba will proceed with selected tasks shown in the schedule (Figure 8-2);
- excavations will not be performed beyond the depths stated in the

- previous sections; and
- the assumptions presented in this Work Plan are considered accurate.

8.3.1 Contingencies and Considerations

Four contingency items have been identified:

- RIDEM Air Discharge Permits;
- RIDEM wetlands permits;
- second excavation required in the Production Area; and
- unforeseen subsurface conditions in the Production Area.

RIDEM Air Discharge Permits

This Work Plan assumes that there will be no Air Discharge Permits or other air permits required from RIDEM or from the local government for soil excavation activities. If mechanical sorting of excavated material is required, the potential for impacting air quality is significant. Both the permitting and the implementation of this activity will represent a significant change in scope and will require revision of the Work Plan, the implementation of the Work Plan, and the Health and Safety Plan, if mechanical sorting is necessary.

RIDEM Wetlands Permits

Portions of the Production Area excavation and the SWMU-5 excavation may be within 200-ft of the Pawtuxet River. Therefore, a RIDEM freshwater wetlands permit or a site remediation exemption (pursuant to Section 6.05 of the RIDEM Freshwater Wetlands Act) may be required. This Work Plan will be submitted to the appropriate agency to determine if the planned activities trigger the permit process. If permits are required, then applications will be prepared and submitted to RIDEM according to the schedule shown in Figure 8-2.

Vertical Limits of Excavation: Production Area

This IRM assumes that there is the potential for a second phase of excavation in the Production Area only, and that the second phase of excavation will be limited in horizontal and vertical extent (i.e. not greater than 2-ft below ground surface). Any deeper excavation could severely impact the program because of the increased potential for additional concrete obstructions and/or abandoned underground utilities.

Production Area Subsurface Conditions

The assumptions of the subsurface conditions in the Production Area are based on

interviews with former employee(s) reportedly present at the time of demolition. There remains a significant degree of uncertainty regarding the depth of subsurface supporting structures for the former buildings. This Work Plan is based on the best available information but cannot address all potential subsurface obstructions. The implementation of this program allows flexibility for the field personnel to make informed decisions regarding the limits of the excavation based on subsurface obstructions.

8.3.2 Critical Success Factors

One critical success factor has been identified:

- Integration of the IRM with stabilization must be well coordinated. The schedule for implementation is presented in Figure 8-2.

Integration of the IRM with Stabilization

The IRM proposed for the Production Area will need to be completed prior to conducting the construction activities proposed for stabilization. Contaminated soil will need to be removed before the soil vapor extraction system can be installed at SWMU-11 and before a parking lot can be constructed on a portion of the Production Area. A schedule which combines conducting the IRM with stabilization is shown in Figure 8-2. In this schedule, field activities proposed for the IRM will be completed before construction for stabilization begins. If unforeseen (or significant) delays in conducting the IRM are encountered, then the schedule for implementing stabilization will be impacted. Every attempt will be made to minimize the routine delays encountered during the implementation of the IRM. Throughout this schedule, weekly monitoring will be performed. As soon as delays are identified, plans to counter them will be proposed and evaluated.

8.4 REPORTING

Within 30 days of receipt of the post-excavation analytical results, a brief letter report containing analytical results and sample locations will be submitted to the USEPA as part of the Monthly Progress Report program already in place.

A final summary report of all IRM activities will be prepared after all data (field, analytical, etc.) have been reviewed and evaluated. This report will include, but not be limited to:

- description of field methods;
- presentation of all post-excavation and waste classification analytical results, including laboratory analytical reports;
- as-built diagrams of the limits of the excavations;

- description of any variances from the Work Plan; and,
- recommendations for additional activities, if warranted.

8.5 SUMMARY

This chapter addressed project management issues for the IRM by updating the Project Management Plan for the RFI. The project direction for this IRM falls under the USEPA and centers on the Ciba Project Coordinator. The IRM is on a separate schedule from all other ongoing activities at the Site. The schedule is organized into six groups of activities:

1. USEPA reviews Work Plan; comments generated by USEPA (if any) are addressed;
2. identify and obtain the necessary permits;
3. prepare the bid specifications and choose a remedial contractor;
4. conduct the pre-mobilization activities (including sampling for waste characteristics);
5. implement the IRM; and
6. prepare reports including the letter report and final IRM activities report.

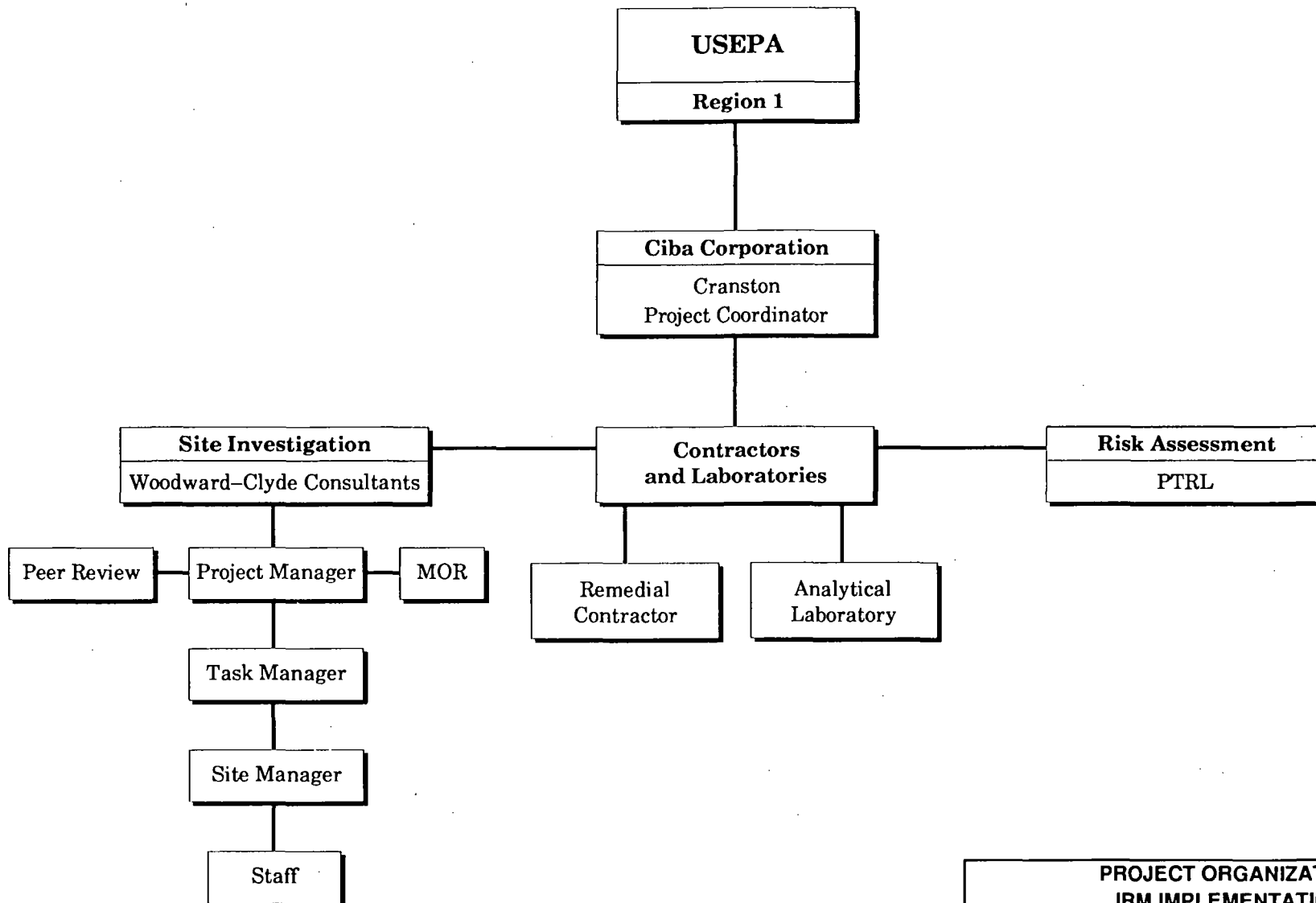
Four contingency/consideration items have been identified:

- RIDEM Air Discharge Permits;
- RIDEM wetlands permits;
- second excavation required in Production Area; and
- unforeseen subsurface conditions encountered in Production Area.

One critical success factor was identified:

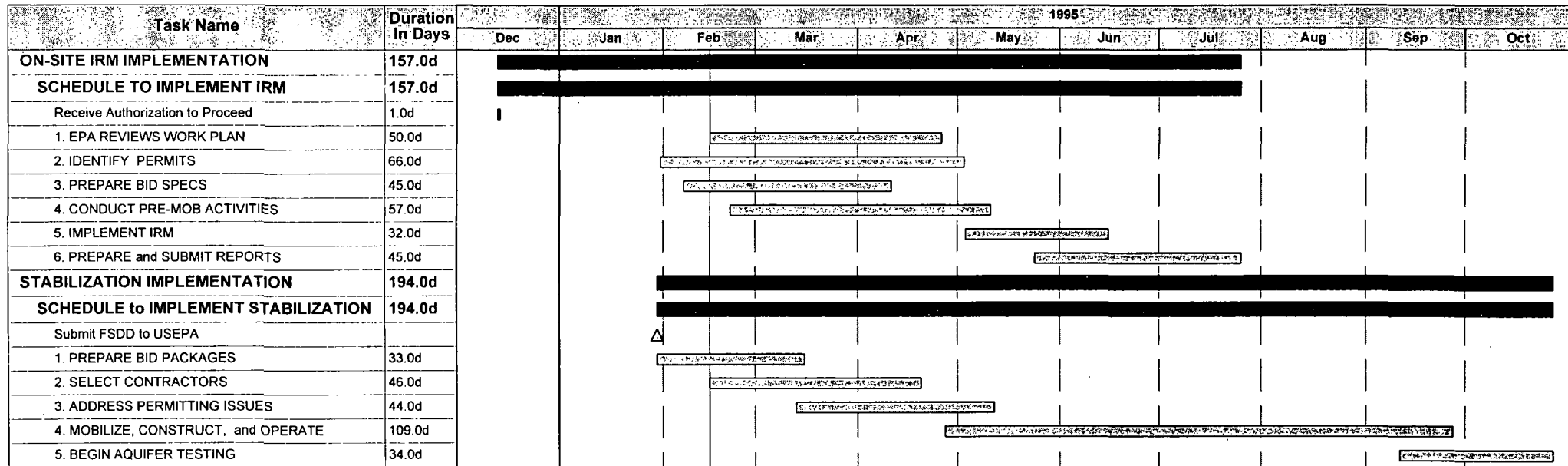
- The integration of the IRM with stabilization must be well coordinated.

Activities performed during the implementation of the soil IRMs will be discussed in the Monthly Progress Reports. A final IRM activities report will be prepared after all the data have been reviewed and evaluated.



PROJECT ORGANIZATION IRM IMPLEMENTATION CIBA-GEIGY FACILITY CRANSTON, RHODE ISLAND			
WOODWARD-CLYDE CONSULTANTS CONSULTING ENGINEERS, GEOLOGISTS AND ENVIRONMENTAL SCIENTISTS WAYNE, NEW JERSEY			
DR. BY:	BAS	SCALE:	NONE
CK'D. BY:	KK	DATE:	30 JAN 1995
PROJ. NO.:	87X4660	FIG. NO.:	8-1

Figure 8-2
Schedule to Implement the On-Site IRM and Stabilization
Ciba Site, Cranston, Rhode Island



Appendix A

**Risk Assessment for the Interim Remediation of the
Production and Warwick Areas at the Ciba
Cranston, Rhode Island Site**

Prepared for:
Ciba-Geigy Corporation
Route 37, West
Toms River, NJ 08754

Prepared by:
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1942 Oak Ridge Turnpike
Oak Ridge, TN 37830

March 9, 1995

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Acronyms and Abbreviated Terms

CMS	Corrective Measures Study
COPC	Chemicals of Potential Concern
CSF	Cancer Slope Factor
CSF _i	Cancer Slope Factor--Inhalation Exposure Route
CSF _o	Cancer Slope Factor--Oral Exposure Route
HEAST	Health Effects Assessment Summary Tables
HHEM	Human Health Evaluation Manual
HI	Hazard Index
HQ	Hazard Quotient
ILCR	Incremental Lifetime Cancer Risk
IN	Level of Intake
IRIS	Integrated Risk Information System
IR _s	Soil Ingestion Rate
LOAEL	Lowest-Observed-Adverse-Effects Level
MPS	Media Protection Standards
NOAEL	No-Observed-Adverse-Effects Level
PAHs	Polycyclic Aromatic Hydrocarbons
PCBs	Polychlorinated Biphenyls
PCDDs	Polychlorinated Dibenzodioxins
PCDFs	Polychlorinated Dibenzofurans
PHERE	Public Health and Environmental Risk Evaluation
QA/QC	Quality Assurance/Quality Control
RCRA	Resource Conservation and Recovery Act
Region I	U.S. Environmental Protection Agency, Region I
RfC	Reference Concentration
RfD	Chronic Reference Dose
Risk Assessment	Risk Assessment for the Interim Remediation of the Production and Warwick Areas
RME	Reasonable Maximum Exposure
Site	Ciba-Geigy's Cranston, Rhode Island Facility
SQL	Sample Quantitation Limit
SRBCs	Sheep Erythrocytes
SWMU	Solid Waste Management Unit

SWMU-5 Solid Waste Management Unit No. 5
THI Total Hazard Index
UCL 95th Percentile Upper Confidence Limit
UR_i Inhalation Unit Risk
USEPA U.S. Environmental Protection Agency
WCC Woodward-Clyde Consultants

Executive Summary

This Risk Assessment was prepared to support the Interim Remedial Measures of the Production and Warwick Areas proposed by Ciba-Geigy Corporation (Ciba) for the Cranston, Rhode Island Site (the Site). It separately evaluates the potential human health risks associated with the Production and Warwick Areas. It is consistent with the approach outlined in the U. S. Environmental Protection Agency's (USEPA) primary risk assessment guidance documents. The Risk Assessment approach and values for exposure assumptions reflect discussions held with the USEPA Region I (Region I) during several meetings and teleconferences, beginning with the May 17, 1994, meeting with Ciba at the Region I offices.

The purpose of the Risk Assessment is threefold:

- Provide estimates of potential risks posed by site-related chemicals in the Production and Warwick Areas of the Site using the conservative guidance specified by Region I.
- Identify the site areas and chemicals that might require corrective action using this risk assessment approach.
- Provide a site-specific risk assessment model using this conservative approach for estimating risk-based Media Protection Standards (MPS) for surface soil.

The Risk Assessment is designed to provide a conservative, quantitative estimate of potential risks associated with residual site-related chemicals in the Production and Warwick Areas. It is based on analytical results from soil samples collected during Phase I and II of the RCRA Facility Investigation field activities. It was performed by identifying chemicals of potential concern (COPC) and carrying them through the risk assessment process. The COPC were determined based on their toxicities, frequencies of detection, and concentrations in site soil.

Regarding future land use, separate exposure scenarios were evaluated for the Production and Warwick Areas. Based on a proposal to use the Production Area as a vehicle parking facility, the Risk Assessment reflects an on-site worker scenario for this area. Unrestricted residential land use was assumed for the Warwick Area.

Results of the Risk Assessment are expressed in terms of potential noncancer health effects and potential cancer risks which are summarized in Figures ES-1 and ES-2. The total hazard index (THI) represents the overall estimated noncancer risks for a given exposure scenario. The potential noncancer risk represented by the THI is considered of no significance if it is equal to or below a

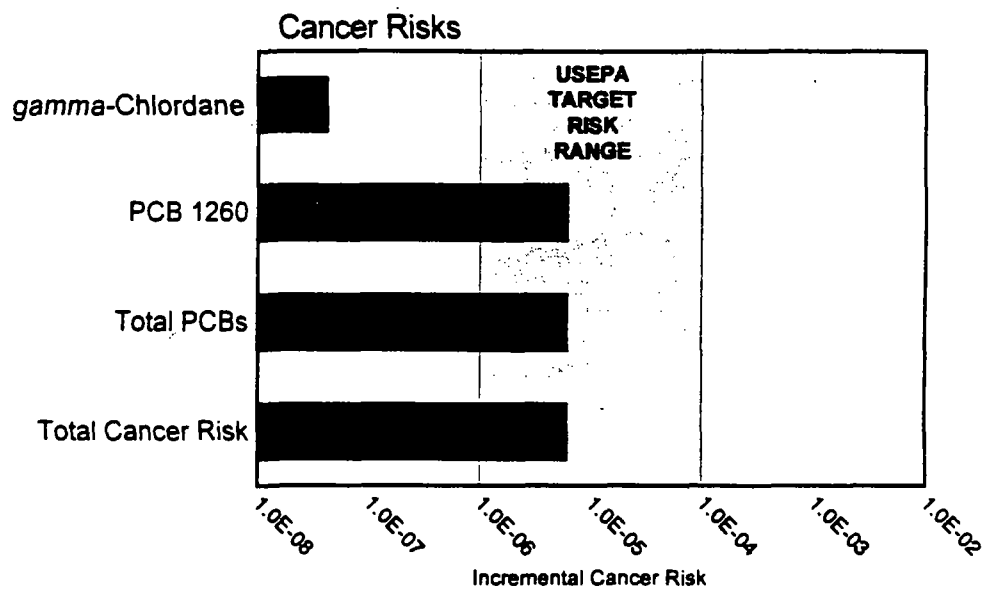
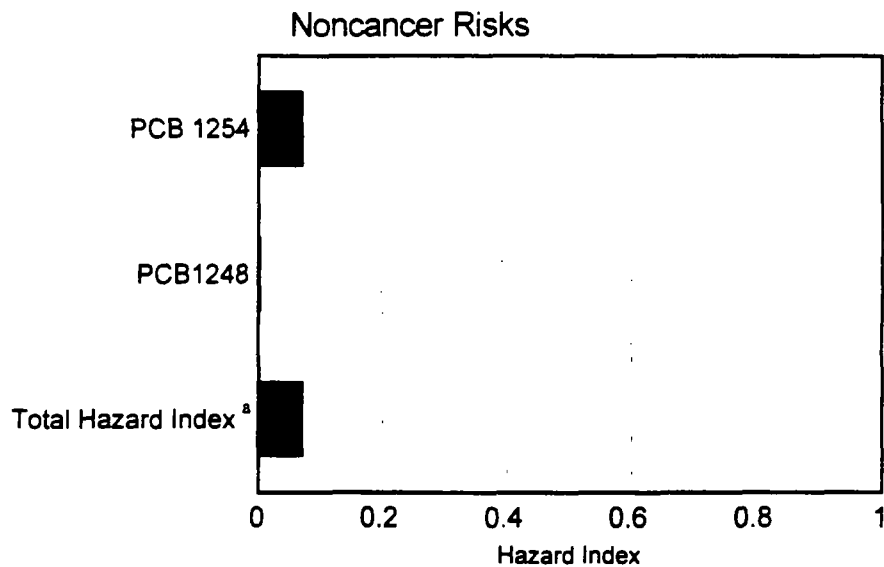
value of 1, and is a potential concern if it is greater than a value of 1 (rounded to a whole number). The potential cancer risk posed is expressed in terms of an incremental lifetime cancer risk (ILCR). The ILCR is an increased probability of cancer above that which exists as "background" (3 out of 10 people) for the general population. The USEPA regards an ILCR of between 1×10^{-6} (1 in 1,000,000) and 1×10^{-4} (1 in 10,000) as acceptable. Thus, this may be interpreted as an increase in the United States baseline cancer incidence from 300,000 per million population to a range of 300,001 to 300,100 per million population. If the ILCR exceeds the upper bound of the target risk range (1×10^{-4}), then further evaluation or corrective action may be indicated.

As shown in Figures ES-1 and ES-2, neither the Production Area nor the Warwick Area are predicted to pose an unacceptable potential risk. The risk numbers presented are highly conservative and may exaggerate actual risks due to a number of factors. For example, the sampling approach was biased in that the field investigation targeted highly localized areas of suspected contamination. Additionally, at Region I's request, the total PCB carcinogenic risk is based on the assumption that all PCBs, including those that are noncarcinogenic (e.g. PCB 1248 and 1254) have a cancer potency factor equal to PCB 1260. These factors are especially significant for the Warwick area, where contamination (PCB 1248 and 1254) is highly localized and no PCB 1260 was detected. From a land-use standpoint, the likelihood of PCB exposure through surface soil is highly unlikely in the Production Area, since the proposed land use is a paved parking facility.

Even with the high degree of conservatism, the Risk Assessment showed that corrective actions are not necessary for the Production and Warwick Areas solely on the basis of potential risk to public health. However, it may be desirable to conduct some limited remediation in these areas for reasons other than potential risk, such as facilitating the productive use of these areas. Based on the concentration and frequency of detection in surface soil (the predominant exposure source), it was determined that PCB removal in the Production and Warwick Areas would provide the greatest benefit in potential risk reduction. Therefore, proposed surface soil MPS values are limited to PCBs only.

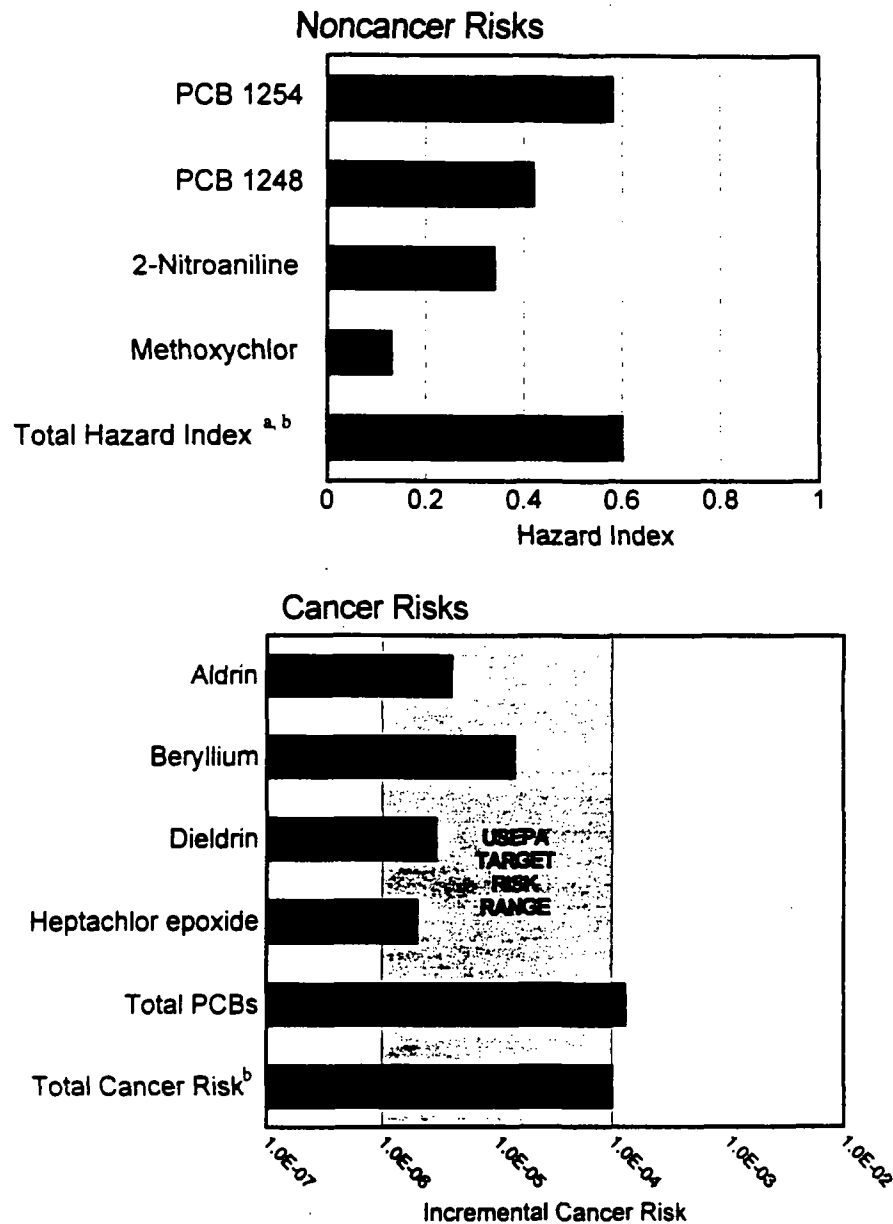
The risk assessment models for the scenarios evaluated were used to estimate risk-based MPS values for total PCBs. Using a THI value of 1, MPSs were back-calculated through the risk assessment model to the respective surface soil concentrations. The resulting total PCBs MPSs are 50 ppm for the Production Area and 9 ppm for the Warwick Area. A clean-up level of 45 ppm (5 ppm lower than that allowed by the risk-based MPS) will be targeted for the

Production Area to ensure that the average residual PCB concentration is below the 50 ppm limit. Based on draft USEPA guidance (Disposal of Polychlorinated Biphenyls; Proposed Rule 12/12/94), the decision was made to reduce the target clean-up level in the Warwick Area to 1 ppm to allow for unrestricted use.



^a Only similar hazards are summed. Refer to Section A7.0 and Attachment 6.

Figure ES-1. Risk Summary for Production Area On-Site Worker Scenario



^a Only similar hazards are summed. Refer to Section A7.0 and Attachment 6.

^b Rounded to one significant figure as described in the Human Health Evaluation Manual (USEPA, 1989).

Figure ES-2. Risk Summary for Warwick Area On-Site Resident Scenario



Section A1

Introduction

A1.0 Introduction

This Risk Assessment was prepared to support the Interim Remediation of the Production and Warwick Areas proposed by Ciba-Geigy Corporation (Ciba) for the Cranston, Rhode Island Site (the Site). It separately evaluates the potential human health risks associated with the Production and Warwick Areas. Figure A1-1 shows the various areas of the Site. This Risk Assessment is limited in scope to those areas targeted for proposed interim remedial measures and is not intended to take the place of the Public Health and Environmental Risk Evaluation (PHERE) required by the Consent Order for the RCRA Facility Investigation of the Site. The Risk Assessment is consistent with the approach outlined in the *U. S. Environmental Protection Agency (USEPA) Risk Assessment Guidance for Superfund, Human Health Evaluation Manual (HHEM)* (USEPA, 1989a). The Risk Assessment approach and values for exposure assumptions include those discussed during several meetings and teleconferences with the USEPA Region I (Region I) beginning with a meeting on May 17, 1994. Topics pertaining only to potential human health risks associated with occupational and residential land-use scenarios are addressed.

A1.1 Purpose and Scope

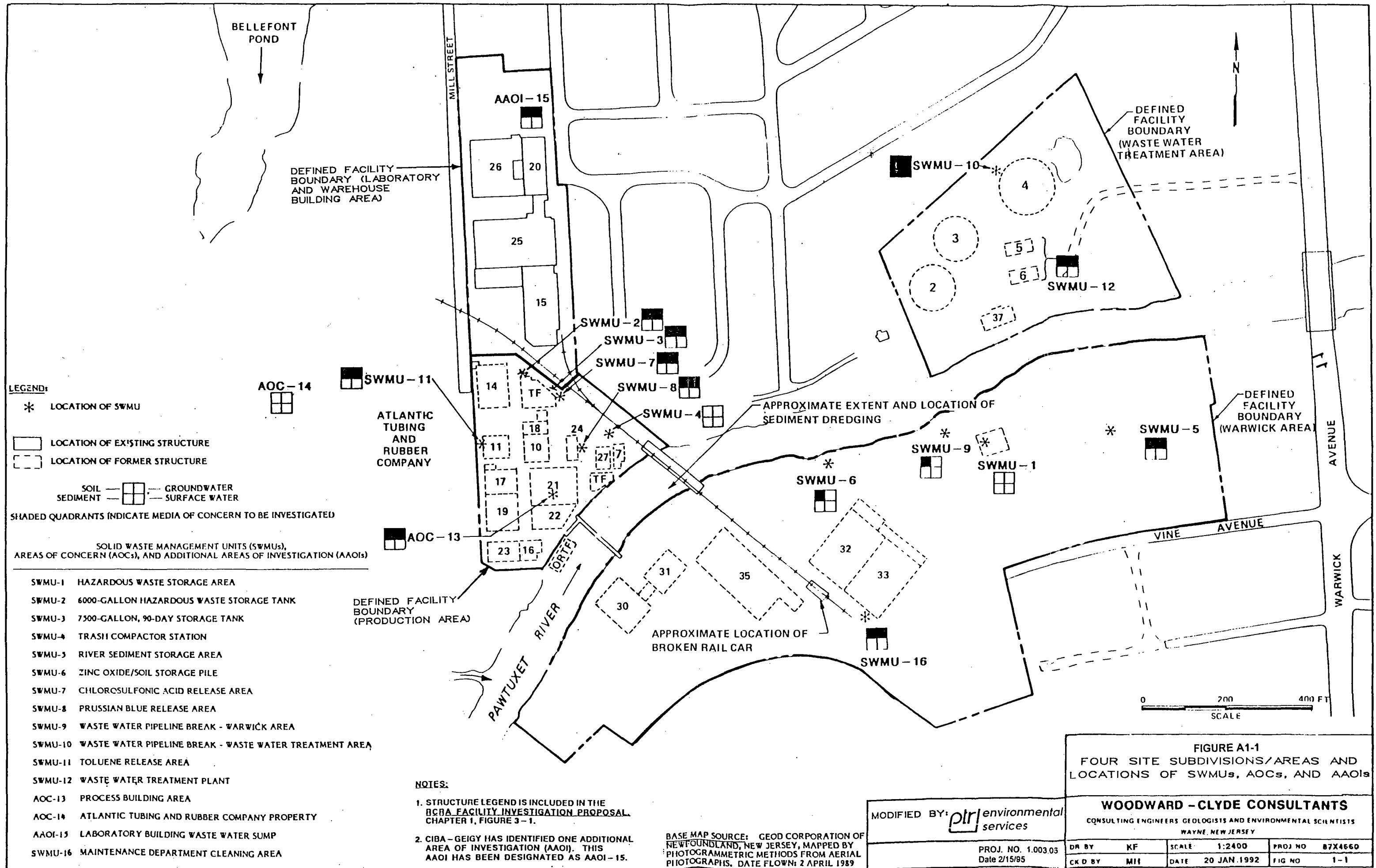
The purpose of the Risk Assessment is threefold:

- Provide estimates of potential risks posed by site-related chemicals in the Production and Warwick Areas of the Site using the conservative guidance specified by Region I.
- Identify the site areas and chemicals that might require corrective action using this Risk Assessment approach.
- Provide a site-specific risk assessment model using this conservative approach for estimating risk-based media protection standards (MPS) for surface soil.

This Risk Assessment is designed to provide a conservative, quantitative estimate of potential risks associated with residual, site-related chemicals in the Production and Warwick Areas. It was performed by selecting chemicals of potential concern (COPC) and carrying them through the risk assessment process consistent with the principals in the HHEM. The COPC were selected based on their toxicities, frequencies of detection, and the concentrations at which they were detected in site soil. Regarding future land use, separate exposure scenarios were evaluated for the Production and Warwick Areas. Based on a proposal to use the Production Area as a vehicle parking facility, the Risk Assessment reflects an on-site worker scenario for this area. Unrestricted residential land use was assumed for the Warwick Area.

A1.2 Report Organization

Section A2.0 describes the risk assessment methods and chemical analytical data on which the Risk Assessment is based. Section A3.0 describes background soil concentrations of chemicals, compares them to on-site concentrations, and defines under what conditions chemicals were eliminated from further consideration in the Risk Assessment. Section A4.0 discusses the COPC selection process and lists the COPC for the two site areas. The exposure assessment, which includes a description of the exposure setting, potential exposure pathways, potential human receptors, chemical intake assumptions, and potential exposure point concentrations, comprises Section A5.0. The toxicity assessment (Section A6.0) describes the cancer and noncancer effects of the COPC. The risk characterization (Section A7.0) discusses the estimated potential cancer risks and noncancer hazards associated with the two site areas. The uncertainties associated with the Risk Assessment are described in Section A8.0. Media Protection Standards are proposed in Section A9.0. References follow the body of the text in Section A10. Tables and figures follow each section of the text. Attachments 1 through 6 provide back-up for the text.



Section A2

Risk Assessment Methods and Analytical Data

A2.0 Risk Assessment Methods and Analytical Data

A2.1 Risk Assessment Methods

The Risk Assessment was performed following HHEM guidance. This includes appropriate use of the validated data, selection of compounds of potential concern (COPC), exposure assessment methodology, toxicity assessment, risk characterization, and uncertainties analysis. The Risk Assessment pertains to interim remediation of soils in the Production and Warwick Areas and has a more limited focus than a typical baseline risk assessment.

The following is a partial list of guidance documents and other sources of information used in the preparation of the Risk Assessment:

- Integrated Risk Information System (IRIS), Toxicology Data Network, National Library of Medicine, final on-line search performed January, 1995.
- *Health Effects Assessment Summary Tables*, Office of Solid Waste and Emergency Response, Washington, D.C., (EPA/540/R-94/020), USEPA, 1994.
- *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons*, Environmental Criteria and Assessment Office, Cincinnati, Ohio (EPA/600/R-93/089), USEPA, 1993.
- *Dermal Exposure Assessment: Principles and Applications*, Interim Report, Office of Research and Development, Washington, D.C., (EPA/600/8-91/011B), USEPA, 1992.
- *Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual, Supplemental Guidance, Standard Default Exposure Factors*, Interim Final, Office of Solid Waste and Emergency Response, Washington, D.C., (OSWER Directive 9285.6-03), USEPA, 1991.
- *Human Health Evaluation Manual, Part B: Development of Risk-Based Preliminary Remediation Goals*, Office of Solid Waste and Emergency Response, Washington, D.C., (OSWER Directive 9285.7-01B), USEPA, 1991.

- *Exposure Factors Handbook*, Office of Health and Environmental Assessment, Washington, D.C., (EPA/600/8-89/043), USEPA, 1990.
- *"Corrective Action for Solid Waste Management Units (SWMUs) at Hazardous Waste Management Facilities, Proposed Rule,"* 55 *Federal Register* 30798, July 27, 1990 USEPA, 1990.
- *Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual, Part A*, Interim Final, Office of Emergency and Remedial Response, Washington, D.C., (EPA/540/1-89/002), USEPA, 1989.
- Region I guidance for oral absorption and dermal absorption of PCBs, internal memo, based on studies performed by Fries et.al., 1989, USEPA, 1995a.
- Region I policy for potential cancer risks related to PCBs, USEPA, 1995b.

Other sources of information were used as needed.

A2.2 Analytical Data

The Risk Assessment is based on analytical results of soil samples provided in electronic database format by Woodward-Clyde Consultants (WCC). These include Phase I and Phase II investigation data. Attachment I provides an evaluation in tabular form of the chemicals detected in Production Area and Warwick Area soils, which includes their detection frequencies and maximum, minimum, mean, and 95th percentile upper confidence limits (UCLs) of the mean concentrations.

Soil samples were designated by WCC as "surface soil" or "soil boring". The surface soil samples were collected at a depth range of 0.5 to 2.0 feet (or an interval within this range). The boring samples were collected in 2-foot intervals from the surface using split-spoon samplers. Because the uppermost boring samples were collected at the 0- to 2.0-foot depth, these are included as surface soil samples in the Risk Assessment. The remaining boring samples are referred to in the Risk Assessment as "subsurface soil".

Surface and subsurface soil samples for the Production Area and Warwick Area were collected in two phases of field investigation, with two rounds of soil sampling in each phase. Phase I-Round 1

sampling took place during November and December, 1990; Phase I-Round 2 during March, 1991; Phase II-Round 1 during July and August, 1993; and Phase II-Round 2 during May, 1994.

Additional surface soil samples were collected from the Production Area in April 1992; these are included as Phase II-Round 1 samples. Sampling locations and analytical methods are identified and discussed in the *RCRA Facility Investigation Interim Report* (Ciba-Geigy, 1991). The sampling program used a biased approach in that specific locations within Site areas suspected of potential contamination were targeted. This is especially true in the Warwick Area which was not used in the daily operations of the Facility. Therefore, the sampling analytical results are not representative of the entire Warwick Area, but predominantly represent only the highly localized area of SWMU-5 (Figure A1-1).

The validated data from each of these sampling rounds were used in the Risk Assessment. Due to quality assurance/quality control (QA/QC) issues reported by WCC, the analytical results for polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) in Rounds 1 and 2 of Phase I were not used in the Risk Assessment. Potential risks associated with PCDDs and PCDFs were evaluated using the Phase II analytical results.

The data evaluations included in Attachment 1 are for the following four data sets:

- Production Area surface soil;
- Production Area combined surface and subsurface soil ("combined soil");
- Warwick Area surface soil; and
- Warwick Area combined soil.

Surface soil and subsurface soil data sets are combined for the soil-to-air transport model used in the exposure assessment (Section A5.0) for volatile chemicals.

A2.2.1 Production Area

Production Area soil samples collected during Rounds 1 and 2 of the Phase I investigation were analyzed for the complete list of Appendix IX parameters, as were some of the samples collected during Round 1 of the Phase II investigation. These came to a total of more than 40 surface soil and 40 subsurface soil samples.

Fifty additional Phase II-Round 1 surface soil samples were collected in April 1992 using a grid sampling pattern. These were analyzed for PCBs only. Only PCB 1248 and PCB 1254 were

detected in these samples. The other PCB mixtures were either not detected in these samples or the data were rejected during data validation.

Ten surface soil samples were collected from the Production Area during Phase II-Round 2. Nine of these samples were analyzed for PCBs only; the tenth was analyzed for PCBs and arsenic. The sample analyzed for arsenic was collected from the same sampling location (SF-A13-C27(S)) as was a Phase I-Round 2 sample in which arsenic was detected at a relatively high concentration (125 mg/kg). The Phase II-Round 2 sample collected from this location was analyzed for arsenic to verify the value found in the sample collected during Phase I-Round 2.

A2.2.2 Warwick Area

Surface and subsurface soil samples were collected in the Warwick Area during Rounds 1 and 2 of Phase I and Round 1 of Phase II. Most of these were analyzed for the complete list of Appendix IX parameters, although some samples were limited to a partial list of Appendix IX. A total of over 30 surface and 20 subsurface soil samples from the Warwick Area were collected for some level of Appendix IX analyses. No Warwick Area soil samples were collected during Phase II-Round 2.

A2.2.3 Background Data

A total of 17 soil samples, 12 surface and 5 subsurface, were collected from background sampling locations. These samples were collected from off-site areas near the Site but not believed to be impacted by the Site. The analytical results of these samples provide baseline concentrations of the local soils. The background surface soil samples were collected during Rounds 1 and 2 of Phase 1, and Round 1 of Phase 2. The subsurface background soil samples were only collected during Phase II-Round 1.

Section A3

On-Site and Background Chemical Concentration Comparison

A3.0 On-Site and Background Chemical Concentration Comparison

The purpose of the Risk Assessment is to evaluate the potential risks associated with chemicals related to past Site activities. Although naturally occurring and miscellaneous chemicals originating from human sources not related to Site activities may also pose potential human health risks, evaluation of risks associated with background soil levels of chemicals in this part of Rhode Island is beyond the scope of the Risk Assessment.

Inorganics are ubiquitous in the environment and were found at detectable concentrations in background soils. Therefore, the concentrations of all the inorganics analyzed for in on-site surface soil were compared to those of near-site background surface soils. This was done by comparing the mean concentration of each inorganic detected in on-site soil to the 95th percentile upper confidence limit (UCL) of the mean concentration of this inorganic in near-site background soils. If the mean concentration of a given inorganic detected in the on-site soil exceeds the UCL of the mean concentration at which that inorganic was detected in near-site background soil, then the inorganic was evaluated in the next step of the Risk Assessment, the selection of chemicals of potential concern (COPC) (Section A4.0 and Attachment 2). If the UCL of the mean concentration of the inorganic chemical is less than or equal to the mean concentration of the near-site background samples, then the inorganic was eliminated from further evaluation in the Risk Assessment. It is noted that because the most likely human receptors would not be exposed to subsurface soils and subsurface inorganics do not volatilize to the surface, only surface soil concentrations were considered in the comparison of on-site inorganics to near-site background inorganics.

The concentrations of organic compounds in near-site background soils were generally assumed to be zero. However, concentrations of polycyclic aromatic hydrocarbons (PAHs) in near-site background samples were observed to approximate those of the on-site samples. PAHs result from all types of combustion and, like inorganics, are ubiquitous in the environment. Thus, PAH concentrations in on-site soils were compared to near-site background concentrations using the same approach as described above for inorganics. The PAHs were detected at higher concentrations and at greater frequencies in both on-site and near-site background surface soils than in subsurface soils. Since exposure at the Site is mostly associated with surface soil (Section A6.0), only surface soil samples were included in this comparison.

The following subsections describe comparisons of Production Area and Warwick Area surface soil concentrations of inorganics and PAHs to those of near-site background samples. PAHs are discussed with respect to both total PAHs and benzo(a)pyrene. Benzo(a)pyrene was selected for particular discussion because it is regarded (along with dibenz(a,h)anthracene) as being among the most potent PAH carcinogens, and it is one of the more commonly detected PAHs in on-site and near-site soils. Data relating to on-site and near-site surface soil concentrations of each detected PAH compound and total PAHs are summarized in Tables A3-1 and A3-2.

Concentrations of the individual PAHs found in urban soils, as available in literature sources, are also shown in these tables.

Based on Site history, neither inorganics nor PAHs were used or produced at the Site. Those detected at concentrations and/or frequencies equal to or less than those found in near-site background soil samples were eliminated from evaluation of potential risks. When an inorganic or PAH was detected in the background samples, and at greater-than-background concentrations on-site, the contribution of background to the on-site concentration was not subtracted from the concentration used in the Risk Assessment.

A3.1 Production Area

A3.1.1 Inorganics

Eight of the 22 inorganics detected in Production Area surface soil exceed their respective concentrations detected in near-site background soils (refer to Attachment 2, Table A2-1). The inorganics exceeding background are cadmium, calcium, copper, magnesium, mercury, nickel, potassium, and zinc. Only these eight inorganics were further evaluated in the Risk Assessment.

Of the eight inorganics detected at above-background concentrations, only calcium and zinc were detected in Production Area surface soil at mean concentrations (20,713 and 184 mg/kg, respectively) exceeding the UCL of the mean background soil concentrations by a factor of two or more. This indicates that six of the eight inorganics detected at higher-than-background concentrations were not greatly above background. The UCL of the mean background concentration for calcium is 1,142 mg/kg and for zinc is 76 mg/kg. The mean zinc concentration of the Production Area surface soil (184 mg/kg) is less than the maximum zinc concentration (219 mg/kg) detected in near-site background surface soil. Although calcium is detected at a much higher concentration in Production Area soils than in background, calcium is a human

macronutrient with very low toxicity. Calcium is commonly found in some natural soils at concentrations up to 400,000 mg/kg (Dragun, 1988), 20 times greater than the mean concentration found in Production Area surface soils.

A3.1.2 PAHs

A3.1.2.1 Total PAHs

Seventeen PAHs were detected in Production Area surface soil, as well as in the near-site background soil. Generally, the frequencies of detection are slightly greater in the background samples than in the Production Area samples (Table A3-1). Six PAHs were detected in the two sample sets at virtually the same frequency, ten were more frequently detected in the background samples, and only benzo(a)anthracene was more frequently detected in the Production Area samples. The two data sets are strikingly similar with regard to those of the 17 PAHs most frequently and least frequently detected. This is illustrated below (frequencies of detection are shown in parentheses):

Most Frequently Detected:

Frequency Rank	Production Area	Frequency Rank	Background
1	Fluoranthene (81%)	1	Fluoranthene (100%)
2	Pyrene (78%)	1	Pyrene (100%)
3	Benzo(b)fluoranthene (73%)	3	Phenanthrene (92%)
4	Chrysene (68%)	4	Chrysene (75%)
4	Phenanthrene (68%)	4	Benzo(b)fluoranthene (75%)

Least Frequently Detected:

Frequency Rank	Production Area	Frequency Rank	Background
17	2-Methylnaphthalene (9.8%)	17	2-Methylnaphthalene (17%)
16	Acenaphthene (12%)	16	Dibenz(a,h)anthracene (25%)
14	Dibenz(a,h)anthracene (24%)	14	Naphthalene (33%)
14	Acenaphthylene (24%)	14	Acenaphthylene (33%)

The five most commonly detected PAHs in Production Area surface soil are also the five most commonly detected in near-site background surface soil. Likewise, three of the four least commonly detected PAHs in the background surface soil are also three of the four least commonly detected in Production Area surface soil. The nearly identical relative concentrations of PAHs detected in on-site and near-site soils strongly suggest that PAHs detected in the Production Area and near-site areas originate from off-site sources unrelated to Site activities. The analytical results discussed above also indicate that PAHs are found ubiquitously in this urban region of Rhode Island.

Table A3-1 also lists background concentrations of PAHs in urban soil that are published in the literature. None of the mean or UCL of the mean concentrations exceed these ranges, and are considerably less than the maximum values of the ranges given. This indicates that the PAH concentrations of Production Area and near-site background surface soil are not higher than expected for an urban setting.

A3.1.2.2 Benzo(a)pyrene

Benzo(a)pyrene was detected at virtually the same frequencies in Production Area (66%) and near-site background surface soil (67%) (refer to Table A3-1). The mean concentration (1.3 mg/kg) found in Production Area surface soil samples is less than the mean concentration (2.6 mg/kg) detected in the background soil samples. Thus, benzo(a)pyrene is found in Production Area surface soil samples at concentrations equal to or less than near-site background.

Comparisons were also made to background soil levels of individual PAHs reported in the literature. White and Vanderslice (1980) list a typical range of 50 to 75 mg/kg for benzo(a) pyrene in urban soil. The concentrations of benzo(a)pyrene detected in both Production Area and near-site background soil are below this range.

It should be noted that the mean concentration for benzo(a)pyrene found in near-site background soil is skewed higher due to one surface soil sample in which this compound was detected at 22 mg/kg. However, even this value is low in comparison to the typical soil concentration range for benzo(a)pyrene (50 to 75 mg/kg) described in the literature.

A3.2 Warwick Area

A3.2.1 Inorganics

Twelve of the 21 inorganics detected in Warwick Area surface soils exceed their respective near-site background soil levels (refer to Attachment 2, Table A2-4). The inorganics which exceed background are antimony, barium, beryllium, cadmium, calcium, chromium, cobalt, copper, cyanide, nickel, potassium, and zinc. These 12 inorganics were carried through to the next step of the Risk Assessment, the COPC selection process (Section A4.0). Six of these inorganics have mean soil concentrations which exceed the UCL of the mean background surface soil concentration by less than a factor of two; these are regarded as slightly above background. The six inorganics exceeding background by more than a factor of two are antimony, cadmium, chromium, copper, nickel, and zinc (refer to Attachment 1, Tables A1-3 and A1-5). Zinc is the only inorganic detected in Warwick Area surface soil at a concentration (2,540 mg/kg) greater than the concentration range (10 to 300 mg/kg) listed in literature sources for typical natural soils (Dragun, 1988; Levinson, 1980).

A3.2.2 PAHs

A3.2.2.1 Total PAHs

Seventeen PAHs were detected in Warwick Area surface soil, as well as in the near-site background soil (Table A3-2). The detection frequency for each compound is greater in background than in Warwick Area surface soil. Relative detection frequencies of PAHs within the Warwick Area soil data set mirror those of the near-site background data set. This observation was also made for the Production Area (Section A3.2.1). The individual PAHs detected most and least frequently in the two data sets are listed below in order of rank with respect to frequency of detection (frequencies of detection are shown in parentheses):

Most Frequently Detected:

Frequency Rank	Production Area	Frequency Rank	Background
1	Pyrene (58%)	1	Pyrene (100%)
2	Fluoranthene (55%)	1	Fluoranthene (100%)
2	Phenanthrene (55%)	2	Phenanthrene (92%)

Least Frequently Detected:

Frequency Rank	Production Area	Frequency Rank	Background
15	Acenaphthene (9.7%)	17	2-Methylnaphthalene (17%)
15	Dibenz(a,h)anthracene (9.7%)	16	Dibenz(a,h)anthracene (25%)
15	Acenaphthylene (9.7%)	14	Acenaphthylene (33%)
14	2-Methylnaphthalene (19%)	14	Naphthalene (33%)

The three most commonly detected PAHs in Warwick Area surface soil are the same, in order, as the three detected most commonly in the near-site background surface soil. Likewise, three of the four least commonly detected PAHs in Warwick Area surface soil are the same as those detected in background soil. Just as for the Production Area (Section A3.1.2.1), the nearly identical relative concentrations of PAHs detected in on-site and near-site soils strongly suggest that PAHs detected in the Warwick Area and near-site areas originate from off-site sources unrelated to Site activities. The analytical results discussed above also indicate that PAHs are found ubiquitously in this urban region of Rhode Island

Table A3-2 also lists background concentrations of PAHs in urban soil that are published in the literature. None of the mean or UCL of the mean concentrations exceed these ranges, and are considerably less than the maximum values of the ranges given. This indicates that the PAH concentrations of Warwick Area and near-site background soil are not higher than expected for an urban setting.

A3.2.2.2 Benzo(a)pyrene

Benzo(a)pyrene was detected at a lower frequency in Warwick Area surface soil (42%) than in near-site background surface soil (67%) (see Table A3-2). The mean concentration (1.2 mg/kg) found in Warwick Area surface soil samples is less than the mean concentration (2.6 mg/kg) detected in the background soil samples. Thus, benzo(a)pyrene is found in Warwick Area surface soil samples at concentrations equal to or less than near-site background.

Comparisons were also made to background soil levels of individual PAHs reported in the literature. White and Vanderslice (1980) list a typical range of 50 to 75 mg/kg for benzo(a)pyrene in urban soil. The concentrations of benzo(a)pyrene detected in both Warwick Area and near-site background soil are below this range.

It should be noted that the mean concentration for benzo(a)pyrene found in near-site background soil is skewed higher due to one surface soil sample in which this compound was detected at 22 mg/kg. However, even this value is low in comparison to the typical soil concentration range for benzo(a)pyrene (50 to 75 mg/kg) described in the literature.

Table A3-1

Comparison of Production Area Polycyclic Aromatic Hydrocarbon (PAH) Concentrations in Surface Soil with those of Near-Site Background Surface Soil

Chemical	Production Area						Near-Site Background						Urban Background
	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL * (mg/kg)	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL* (mg/kg)	Typical Concentration (mg/kg)
2- Methylnaphthalene	4/41	9.8	0.038	0.38	1.3	1.7	2/12	17	0.57	4.5	0.66	1.3	NA ^b
Acenaphthene	10/41	24	0.057	0.21	1.2	1.7	5/12	42	0.031	5.4	0.69	1.5	NA
Acenaphthylene	5/41	12	0.043	0.18	1.3	1.7	4/12	33	0.044	0.61	0.30	0.4	NA
Anthracene	24/41	59	0.034	1.6	0.88	1.3	7/12	58	0.041	20	2.2	5.1	NA
Benzo(a)anthracene	28/41	68	0.15	3.1	1.1	1.5	7/12	58	0.28	28	3.2	7.4	20 ^c
Benzo(a)pyrene	27/41	66	0.024	3.1	1.3	1.7	8/12	67	0.13	22	2.6	5.8	50-75 ^d
Benzo(b)fluoranthene	30/41	73	0.027	4.3	1.6	2.0	9/12	75	0.026	36	4.2	9.5	NA
Benzo(g,h,i)perylene	21/41	51	0.13	2.9	1.4	1.8	8/12	67	0.080	12	1.6	3.3	100 ^e
Benzo(k)fluoranthene	27/41	66	0.074	5.5	1.5	1.9	8/12	67	0.079	43	4.7	11	NA
Chrysene	28/41	68	0.15	3.3	1.2	1.6	9/12	75	0.14	30	3.4	7.9	20 ^d
Dibenz(a,h)anthracene	10/41	24	0.046	0.68	1.2	1.7	3/12	25	0.12	3.7	0.61	1.1	NA
Fluoranthene	33/41	80	0.051	8.4	1.6	2.1	12/12	100	0.043	57	6.6	15	5-120 ^d
Fluorene	12/41	29	0.048	0.18	1.2	1.7	5/12	42	0.053	9.4	1.1	2.5	NA

Table A3-1, continued

Chemical	Production Area						Near-Site Background						Urban Background
	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL ^a (mg/kg)	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL ^a (mg/kg)	Typical Concentration (mg/kg)
Indeno(1,2,3-cd)pyrene	21/41	51	0.045	2.3	1.3	1.8	7/12	58	0.23	14	1.8	3.9	NA
Naphthalene	14/41	34	0.033	0.68	0.83	1.3	4/12	33	0.023	7.3	0.86	1.9	NA
Phenanthrene	28/41	68	0.093	5.0	1.1	1.5	11/12	92	0.052	69	7.2	17	NA
Pyrene	32/41	78	0.061	6.7	1.8	2.3	12/12	100	0.038	56	6.3	15	5-120 ^d
Total PAHs ^f					22						48		

^a 95th percentile upper confidence limit of the mean concentration.

^b "NA" = Information not available.

^c Source: IRAC, 1973.

^d Source: White and Vanderslice, 1980.

^f The mean concentration for Total PAHs was derived by adding the mean concentration of each individual PAH in the data set.

Table A3-2
Comparison of Warwick Area Polycyclic Aromatic Hydrocarbon (PAH) Concentrations
in Surface Soil with those of Near-Site Background

Chemical	Warwick Area						Near-Site Background						Urban Background
	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL ^a (mg/kg)	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL ^a (mg/kg)	Typical Concentration (mg/kg)
2- Methylanthralene	6/31	19	0.014	0.36	1.3	2.0	2/12	17	0.57	4.5	0.66	1.3	NA ^b
Acenaphthene	3/31	9.7	0.016	0.16	1.4	2.0	5/12	42	0.031	5.4	0.69	1.5	NA
Acenaphthylene	3/31	9.7	0.061	0.11	1.4	2.0	4/12	33	0.044	0.61	0.30	0.40	NA
Anthracene	10/31	32	0.031	0.32	1.3	2.0	7/12	58	0.041	20	2.2	5.1	NA
Benzo(a)anthracene	15/31	48	0.14	1.6	0.97	1.4	7/12	58	0.28	28	3.2	7.4	20 ^c
Benzo(a)pyrene	13/31	42	0.025	1.7	1.2	1.6	8/12	67	0.13	22	2.6	5.8	50-75 ^d
Benzo(b)fluoranthene	14/31	45	0.042	2.8	1.2	1.6	9/12	75	0.026	36	4.2	9.5	NA
Benzo(g,h,i)perylene	9/31	29	0.064	1.2	1.4	2.1	8/12	67	0.08	12	1.6	3.3	100 ^e
Benzo(k)fluoranthene	13/31	42	0.062	3.6	1.3	1.8	8/12	67	0.079	43	4.7	11	NA
Chrysene	14/31	45	0.12	2.3	1.07	1.5	9/12	75	0.14	30	3.4	7.9	20 ^d
Dibenz(a,h)anthracene	3/31	9.7	0.083	0.13	1.4	2.0	3/12	25	0.12	3.7	0.61	1.1	NA
Fluoranthene	17/31	55	0.038	3.7	1.2	1.6	12/12	100	0.043	57	6.6	15	5-120 ^d

Table A3-2, continued

Chemical	Warwick Area						Near-Site Background						Urban Background
	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL ^a (mg/kg)	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL ^a (mg/kg)	Typical Concentration (mg/kg)
Fluorene	7/31	23	0.035	0.23	1.3	2.0	5/12	42	0.053	9.4	1.1	2.5	NA
Indeno(1,2,3-cd)pyrene	8/31	26	0.07	0.86	1.4	2.1	7/12	58	0.23	14	1.8	3.9	NA
Naphthalene	16/31	52	0.036	3.5	1.1	1.6	4/12	33	0.023	7.3	0.86	1.9	NA
Phenanthrene	17/31	55	0.18	1.7	0.87	1.1	11/12	92	0.052	69	7.2	17	NA
Pyrene	18/31	58	0.053	3	1.3	1.6	12/12	100	0.038	56	6.3	15	5-120 ^d
Total PAHs ^f					21						47.9		

^a 95th percentile upper confidence limit of the mean concentration.

^b "NA" = Information not available.

^c Source: IARC, 1973.

^d Source: White and Vanderslice, 1980.

^e Source: USEPA, 1983.

^f The mean concentration for Total PAHs was derived by adding the mean concentration of each individual PAH in the data set.



Section A4

Chemicals of Potential Concern

A4.0 Chemicals of Potential Concern

The COPC were selected using a screening process based on HHEM guidance and detailed in Attachment 2. Detected concentrations, frequencies of detection, and toxicities were considered during screening. Comparisons to background concentrations regarding inorganics and PAHs is described in Section A3.0. PAHs and certain inorganics were previously removed from the selection process discussed here and detailed in Attachment 2. The purpose of using the screening process was to limit the Risk Assessment to the few COPC in each Site area which represent the majority of human health risks. Separate COPC were selected for cancer and noncancer risks. These COPC were carried through the risk assessment process.

The COPC for the respective areas are listed below:

PRODUCTION AREA

Cancer Effects

PCB 1260
gamma-Chlordane

Noncancer Effects

PCB 1248
PCB 1254

WARWICK AREA

Cancer Effects

Aldrin
Beryllium
Dieldrin
Heptachlor epoxide

Noncancer Effects

PCB 1248
PCB 1254
2-Nitroaniline
Methoxychlor

Toxicity information for the COPC is presented in Section A6.0.

Section A5

Exposure Assessment

A5.0 Exposure Assessment

The exposure assessment is a critical component of the human health risk assessment. Exposure assessment methodologies used in the Risk Assessment and the resulting estimated potential exposures are presented in detail in Attachment 3. With respect to chemical hazards, exposure may be defined as the contact of an individual with a chemical agent. Exposure itself does not connote risk, but without exposure or potential exposure a chemical agent poses no hazard or risk.

Exposure assessment in human health risk assessment is used to estimate the quantity of a given chemical that could cross the exchange boundaries between the environment and the body. These boundaries are generally at the gastrointestinal tract, the lungs, and the skin. But before an estimation may be made regarding the quantity of exposure, appropriate scenarios must be developed under which exposure could potentially occur.

The basic steps of an exposure assessment are to:

- Characterize the exposure setting;
- Identify potential exposure pathways;
- Identify human receptors;
- Develop exposure scenarios;
- Develop exposure models; and
- Quantify exposure.

The exposure setting consists of the physical environment, including the proximity of the site to current human populations. The identification of potential exposure pathways considers the characterization of the exposure setting, impacted environmental media, and medium-to-medium transport. The identification of human receptors includes both current and future populations identified during the characterization of the exposure setting. Potential future land uses are evaluated to identify potential future human receptors. Exposure scenarios are developed based on the receptors and potential exposure pathways. Determinations are made regarding which routes of exposure are appropriate for inclusion in the exposure assessment for each identified potential human receptor population. The associated routes of exposure for an identified receptor population is referred to as an exposure scenario.

Exposure quantification uses information from the previous exposure assessment steps. Exposure equations are used to quantify exposure associated with each selected pathway in the exposure

scenario, and these comprise the exposure models. Variables used in the exposure equations include, among others, measured concentrations of chemical in the media, contact rates with the media, frequency of exposure, exposure duration per exposure event, body weight of the exposed individual, total duration over which an individual is exposed, and the time period over which the exposure is averaged. Values for these input variables are site-, medium-, and receptor-specific and may include measured, modeled, or default values.

A5.1 Exposure Setting

The Site is located along the Pawtuxet River in Cranston, Rhode Island. The climate may be characterized as temperate with four well-defined seasons, and is heavily influenced by the Narragansett Bay and the Atlantic Ocean. The mean annual temperature is approximately 50°F, with a daily mean during the coldest month (January) of 29°F and the warmest month (July) of 73°F. The mean annual number of freezing days (minimum temperature of 32°F or less) is 114. The average annual rainfall is approximately 42 inches per year. Measurable precipitation (0.01 inches of rain equivalence) averages 124 days annually and is typically distributed evenly throughout the year. The annual snowfall averages approximately 36 inches, over half of which usually falls during January and February. The wind blows most commonly from a northwestern direction and least commonly from an eastern direction. The average annual wind speed is 11 miles per hour. Meteorological data are from the weather station in Providence, Rhode Island and are contained in Volume 1 of the *RCRA Facility Investigation Proposal* (Ciba, 1990). Additional data are from the National Oceanic and Atmospheric Administration (1990).

The Production Area is located on the north shore of the Pawtuxet River in Cranston; the Warwick Area is on the south shore of the Pawtuxet River and is in Warwick, Rhode Island (Figure A1-1). Areas surrounding the Site are used for commercial, industrial, or residential purposes. The area west of the Production Area is industrial; areas north and east of the Production Area are residential. Based on different levels of impact and probable future land use, the Production Area, as defined in the Consent Order with the USEPA, is for the purpose of the Risk Assessment divided into two parcels: the Laboratory and Warehouse Building Area and the Production Area. These are identified on Figure A1-1. Virtually no site-related chemicals were found in the Laboratory and Warehouse Building Area, and any potential risks will be evaluated separately. Therefore, only the risks associated with the Production Area identified on Figure A1-1 are evaluated in the Risk Assessment.

The Warwick Area is bordered by land in commercial use to the east and residential use to the south. The river lies north and west of this area.

A5.2 Potential Exposure Pathways and Human Receptors

An exposure pathway may be defined as a course that a chemical may take from a source of contamination to an individual. The following four elements are necessary for an exposure pathway to be complete:

- Contamination source and release mechanism;
- Retention medium or transport medium;
- Point of potential human contact with the impacted medium; and
- Human exposure route at the contact point.

The sources and release mechanisms involve previous chemical manufacturing, chemical handling, and waste handling and disposal activities which have occurred in the Site areas. Impacted media which may serve to retain and/or release the contamination are surface soils, subsurface soils, and groundwater. Points of human contact with the impacted media are dependent on land use.

One purpose of the Risk Assessment is to provide conservative estimates of risk. On-site residential use would represent a "worst-case" land use. Ciba has assumed for risk assessment purposes that future on-site residential risk ought to be evaluated for the Warwick Area. Although residential land use may not be the most probable for this area, to be conservative the Risk Assessment evaluates this land use. On-site occupational exposure is assumed for the Production Area because it is being proposed for use by the City of Cranston as parking for city vehicles, and as a storage and loading area for road salt, sand, and snow removal equipment. An on-site worker will potentially occupy this area full-time, but only during the four coldest months of the year performing activities related to snow and ice management of City streets. Parking and vehicle removal by a wide array of City employees will be the only activity for the other 8 months of the year (City of Cranston, 1995). Vehicle maintenance will be conducted at other locations.

These scenarios for the Production and Warwick Areas are evaluated assuming that no modifications are made to the property, such as soil removal or bringing in clean topsoil. The media that may affect a future on-site resident or on-site worker include surface and subsurface soils. Exposure pathways associated with these scenarios are:

- Direct contact with surface soil resulting in incidental ingestion;
- Direct contact with surface soil resulting in dermal absorption;
- Inhalation of airborne chemicals associated with fugitive dust emissions from surface soil; and
- Inhalation of volatilized chemicals associated with surface and subsurface soil.

Figure A5-1 illustrates the exposure pathways evaluated in the Risk Assessment for each exposure scenario. Values for the exposure assumptions for these two scenarios are shown in Table A5-1.

It was determined previously that because municipal water is available and the upgradient shallow groundwater is of poor quality, groundwater from the shallow aquifer underlying the vicinity of the site is not potable. In addition, virtually no site-related contamination was found in deeper aquifers. Site-related chemicals were detected only in shallow groundwater, which follows a strong gradient toward the Pawtuxet River which borders the Site. A RCRA Stabilization Action is addressing this groundwater in the Production Area.

A5.3 Potential Exposure Point Soil Concentrations

The COPC concentrations for the environmental media pertinent to the exposure assessment are shown for the two Site areas in Table A5-2. The concentrations given for surface soils and combined surface and subsurface soils were derived from direct measurements (Attachment 1). Air concentrations shown on Table A5-2 are predicted from measured surface soil and combined soil concentrations using the modeling procedures described in Attachment 4.

Concentrations of COPC for these soils are the lesser of either the 95th percentile UCL of the means or the maximum detected concentrations. For compounds detected in one or more surface soil samples from a given site area, the 95th percentile UCLs of the means were calculated using the detected value, or one-half the sample quantitation limit (SQL) for samples in which the chemical was not detected. Whether the 95th percentile UCL of the arithmetic or geometric mean was used is dependent on whether the data set is best described as a normal or lognormal distribution. The method of evaluation for statistical distribution type is described in Section A5.3.2. The surface soil values in Table A5-2 were used in the soil ingestion and dermal absorption exposures estimated for the on-site residential and worker scenarios (Attachment 3). They were also used in the inhalation pathway exposure estimates of fugitive dust (Attachment 4). The values shown in Table A5-2 for combined subsurface and surface soil concentrations were used for estimating exposure to chemical vapors (Attachment 4).

A5.3.1 Total PCBs Data Sets

Region I policy is to assume that all PCBs have the same cancer potency as PCB 1260 (see Sections A6.0 and A7.0) (USEPA, 1995b). However, soil samples are analyzed and concentrations reported for the separate PCB mixtures. Thus, to assess the potential risks of total PCBs as requested by Region I, the analytical results of all the PCBs detected within a medium were combined to form a separate data set for that medium. For example, three PCBs were detected in Production Area surface soil. The concentrations of PCB 1248, PCB 1254, and PCB 1260 were summed for each Production Area surface soil sample. For samples in which a given PCB was not detected, one-half the sample quantitation limit (SQL) was used. This same approach was taken for Warwick Area surface soil, except that only PCB 1248 and PCB 1254 were summed because neither PCB 1260 nor any other PCB was detected in any Warwick Area surface soil sample. The 95th percentile UCL of the mean was used as the exposure point concentration, just as for the data sets of the respective COPC.

The combined surface soil and subsurface soil data sets are used in the Risk Assessment only for the soil-to-air volatilization model. Because different volatilization rates have been modeled for the individual PCBs, the modeled gaseous concentrations of the separate PCBs were summed to derive an overall exposure to total PCBs with regard to this exposure pathway.

A5.3.2 Statistical Distribution of Chemicals in Soil

Statistical analyses were performed to determine the type of distribution represented by each COPC detected in soil. The type of statistical distribution of the chemical analytical data should be identified, if possible, for a more meaningful exposure point concentration estimate. If a given analyte is detected in too few soil samples, then the type of statistical distribution of the analyte in the soil cannot be reliably ascertained. As described in Section A5.3, if a chemical is not detected in a given sample, then one-half the SQL is the assumed concentration. These one-half SQL values do not accurately portray the actual concentrations, but are used expressly for exposure assessment purposes. Thus, if a chemical is detected too infrequently, the statistical distribution of the chemical in Site soils cannot be reliably identified. This is particularly true if SQL values of the nondetected samples are high relative to the detected values in a data set. The statistical methods and description of the general procedures used in the Risk Assessment to determine distribution type are described in the following paragraphs.

Each data set was first evaluated for frequency of detection. The Risk Assessment uses a lower limit detection frequency of 75% to determine whether a data set can be statistically tested to

evaluate its distribution. The distribution of chemicals detected at frequencies of 75% or greater are evaluated using statistical tests for departures from lognormality and normality that are based on skewness and kurtosis (Bowman and Shenton, 1975). If the data set meets the test criteria for lognormality, then the chemical is assumed to be lognormally distributed over the given Site area, and the 95th percentile UCL of the geometric mean is used to estimate the exposure point concentration. If the data set fails the test for lognormality, then a test for departures from normality is performed. If the data meet the criteria for normality, then the chemical is assumed to be normally distributed over the given Site area, and the 95th percentile UCL of the arithmetic mean concentration is used to estimate the exposure point concentration. If a data set meets the criteria for neither statistical distribution, then the distribution that better fits the data, based on histograms and the results of the respective statistical tests, is the distribution assumed in the Risk Assessment for that data set.

Data sets with detection frequencies of less than 75% were not generally evaluated statistically, but were assumed to be normally distributed, unless otherwise stated (see Section A5.3.2.1). It may be more accurate to assume lognormality for these data sets, because chemical contaminants in soil tend to be lognormally distributed (USEPA, 1991c). But, since the distribution cannot generally be ascertained from these data sets and geometric mean values tend to be less than their corresponding arithmetic mean values, these data sets were generally assumed to be normally distributed as suggested in the HHEM.

A5.3.2.1 PCB 1248

During evaluation of the Production Area data sets for PCB 1248, several observations were made related to its statistical distribution. Although the Production Area soil sample with the highest concentration of PCB 1248 has a reported concentration of 4,500 mg/kg, this value is more than an order of magnitude higher than the next highest reported concentration (430 mg/kg). Further, only 6 of the other 97 samples were found to have a concentration of 7 mg/kg or greater, and approximately 80% of the samples were detected at less than 1 mg/kg; for all nondetects, one-half the SQL was less than 1 mg/kg. With a cursory review, these data appear to indicate a lognormal distribution. Therefore, even though the detection frequency of PCB 1248 in Production Area surface soil is only 39%, the test for lognormality was performed on this data set. Similar observations were made concerning the PCB 1248 combined surface and subsurface soil data set.

The test results for PCB 1248 indicate that both the surface soil and combined soil data sets for

PCB 1248 more closely fit a lognormal than a normal distribution, with the 4,500 mg/kg sample excluded as an outlier. Histograms of the Production Area surface soil (Figure A5-2) showing the concentration frequencies of the raw and log-transformed data for PCB 1248 clearly depict the log-transformed data as more closely following a normal curve, indicating that the set best fits a lognormal distribution. The same observations can be made from the histograms in Figure A5-3 with respect to Production Area combined soil. Therefore, the data for PCB 1248 in the Production Area surface and combined was regarded as lognormal, and the 95th percentile UCL of the geometric mean was used as the exposure point concentration in the Risk Assessment. It is noted that the presence of nondetects may not greatly influence the results of the statistical tests because the SQL values are low in comparison with the detected concentrations; all values of 0.7 mg/kg or greater represent detected concentrations.

A5.3.2.2 PCB 1254

The statistical test results indicate that PCB 1254 is lognormally distributed in both Production Area surface soil and combined soil. That the distribution of PCB 1254 is better described as a lognormal than normal distribution is evident from the histograms shown on Figures A5-4 and A5-5 for Production Area surface and combined soils, respectively. These depict the log-transformed data sets as more closely following a normal curve than do the nontransformed data. Thus, these data sets were regarded as lognormal, and the 95th percentile UCLs of the respective geometric means were used in the Risk Assessment.

A5.3.2.3 Total PCBs

The statistical test results indicate that the total PCBs data sets are lognormally distributed for both the Production Area surface soil and combined soil data sets. The histograms in Figure A5-6 show the log-transformed data for Production Area surface soil more closely following a normal curve than do the nontransformed data. This indicates that the surface soil data set more closely follows a lognormal than a normal distribution. Thus, the total PCBs surface soil data set was regarded as lognormal, and the 95th percentile UCL of the geometric mean was used in the Risk Assessment. As explained in Section A5.3.1, the combined surface and subsurface soil data are used for the modeling of soil-to-air volatilization, and the different PCBs are modeled as having different volatilization rates. Therefore, the sum of the separate gaseous phase air concentrations for PCB 1248, PCB 1254, and PCB 1260 was used in the Risk Assessment for this exposure pathway, instead of basing the air modeling on the UCL of the geometric mean concentration of the total PCBs found in Production Area combined soil.

A5.4 Exposure Assessment Results

The calculated potential exposures are determined by a number of exposure assumptions and variables for each scenario, as presented in Table A5-1, and the results are detailed in Attachment 3.

Table A5-1
Exposure Assumption Values for Residential and On-Site Worker Scenarios

Parameter	Units	Exposure Scenario Values	
		On-Site Worker	On-Site Resident
Body Weight (BW)	kg	70 ^a	70/15 ^b
Averaging Time - Noncarcinogenic (AT _n)	days	9,125 ^c	10,950 ^c
Averaging Time - Carcinogenic (AT _c)	days	27,375 ^d	27,375
Exposure Frequency (EF)	(events/yr)	80 ^a	350 ^f
Conversion Factor (CF)	(kg/mg)	1 x 10 ⁻⁶	1 x 10 ⁻⁶
Exposure Duration (ED)	yr	25 ^a	30 ^h
Soil Ingestion Rate (IR _s)	(mg/day)	50 ⁱ	100/200 ^j
Fraction of Soil Originating from Source (FS)	(none)	1.0 ^k	0.7 ^l
Inhalation Rate (I _n R)	(m ³ /hr)	1.4 ^m	0.6/0.3 ⁿ
Exposure Time (ET)	(hr/day)	8 ^o	16 ^p
Body Surface Area Exposed to Soils (SA _s)	(cm ² /event)	5,000 ^q	2,000/5,000 ^r
Soil Adherence Factor (AF)	(mg/cm ²)	0.5/0.2 ^s	0.5/0.2 ^s

^aDefault value for an adult (USEPA, 1991a).

^bAdult/child default values (USEPA, 1991a).

^cEquals ED x 365 days/yr.

^dEquals a lifetime (75 years x 365 days/yr).

^e85 winter work days/year (17 weeks or about 4 months), minus 5 days vacation and holidays.

^f365 days/year minus 15 vacation days, holidays, weekend trips equals 350 exposure days/year for indoor and outdoor inhalation exposure (USEPA, 1991a). 365 days/year minus 120 winter days/year minus 15 vacations days, holidays, weekend trips equals 230 outdoor exposure days/year for ingestion and inhalation exposures.

^gUpper-bound estimate for time at one place of employment (USEPA, 1991a).

^hUpper-bound estimate for time at one residence (USEPA, 1991b).

ⁱDefault value for industrial/commercial occupations (USEPA, 1991a).

^jDefault value for adult/child residents (USEPA, 1991a).

^kAssumes worker spends all of his/her workday in the parking lot (contaminated area).

^lAssumes a resident spends 8 hours (about 30%) of his/her time away from home.

^mValue for moderate activity (USEPA, 1991b).

ⁿAdult (USEPA, 1990a) child (International Commission on Radiation Protection, 1976).

^oStandard workday.

^pMean hours per day spent at home by men and women is 15.4 (USEPA, 1990a).

^qDefault value, 25% of the total surface area of an average adult (USEPA, 1992), as requested by USEPA Region I.

^rDefault value, 25% of the total surface area of an average adult (USEPA, 1992)/Child as requested by USEPA Region I.

^sAn AF of 0.5 for hands and an AF of 0.2 for the rest of body area assumed to be exposed to soil (USEPA, 1992).

Table A5-2
Potential Upper-Bound Exposure Point Concentrations for Chemicals of
Potential Concern

PRODUCTION AREA

Chemical	Surface Soil ^a (mg/kg)	Combined Soil ^b (mg/kg)	Airborne Emissions ^c (mg/m ³)
PCB-1248	0.44	0.21	3.65×10^{-6}
PCB-1254	3.6	2.0	9.65×10^{-6}
PCB-1260	6.1	6.4	1.85×10^{-7}
<i>gamma</i> -Chlordane	0.13	0.070	2.01×10^{-6}

WARWICK AREA

Chemical	Surface Soil ^a (mg/kg)	Combined Soil ^b (mg/kg)	Airborne Emissions ^c (mg/m ³)
PCB-1248	15	9.7	1.52×10^{-6}
PCB-1254	5.2	3.3	1.43×10^{-7}
2-Nitroaniline	7.0	7.0	1.51×10^{-6}
Methoxychlor	232	199	8.23×10^{-7}
Aldrin	0.21	0.14	3.80×10^{-6}
Beryllium	0.72	0.77	2.92×10^{-9}
Dieldrin	0.16	0.11	3.38×10^{-6}
Heptachlor epoxide	0.19	0.13	1.81×10^{-7}

^aLesser of the 95th percent upper confidence limit (UCL) of the mean concentration or the maximum detected concentration in surface soil samples.

^bLesser of the 95th percent UCL of the mean concentration or the maximum detected concentration in combined surface and subsurface samples.

^cModeled from soil concentrations. Includes fugitive dust emissions predicted using surface soil and gaseous emissions using combined surface and subsurface soil. Refer to Section A5.3.1 of text and Attachment 4.

**Figure A5-1
Exposure Assessment Schematic**

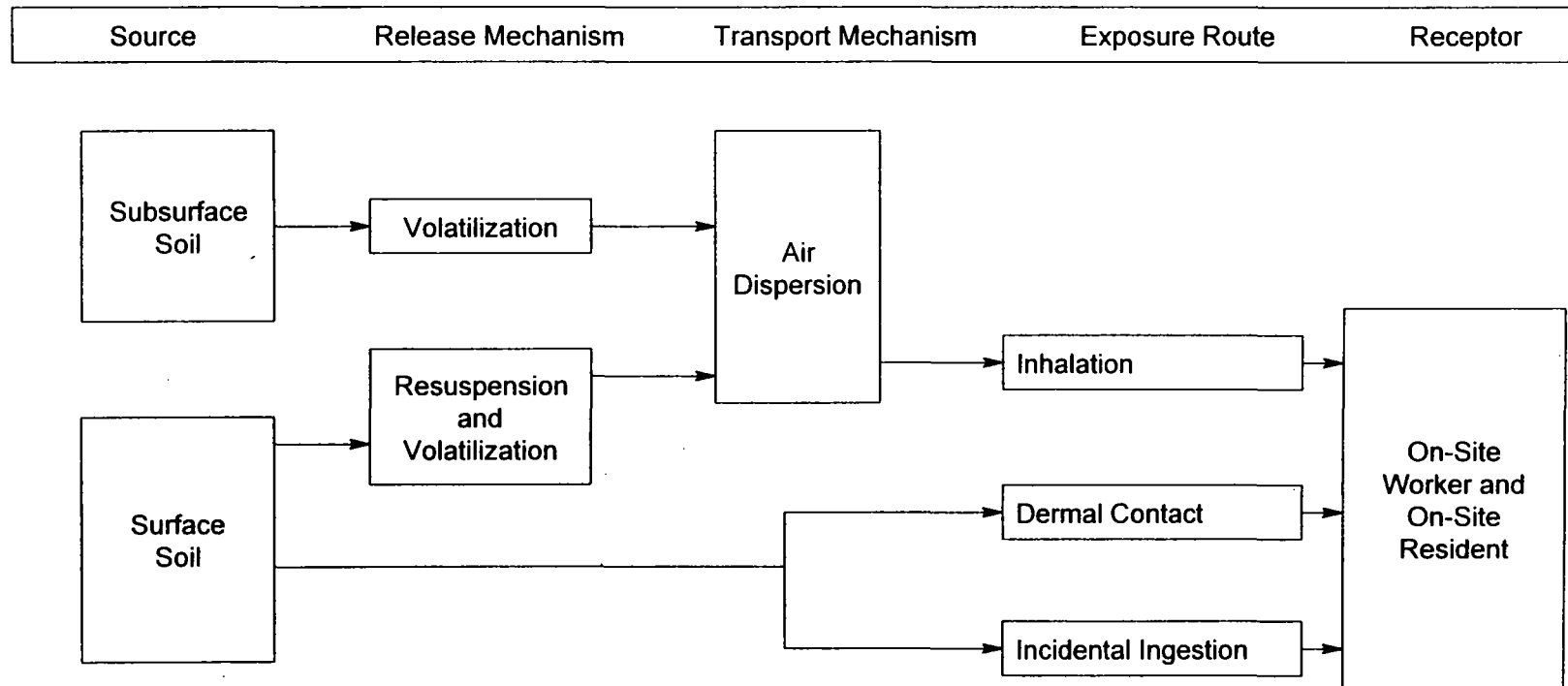


Figure A5-2
Production Area Surface Soil
PCB 1248 Data Distribution

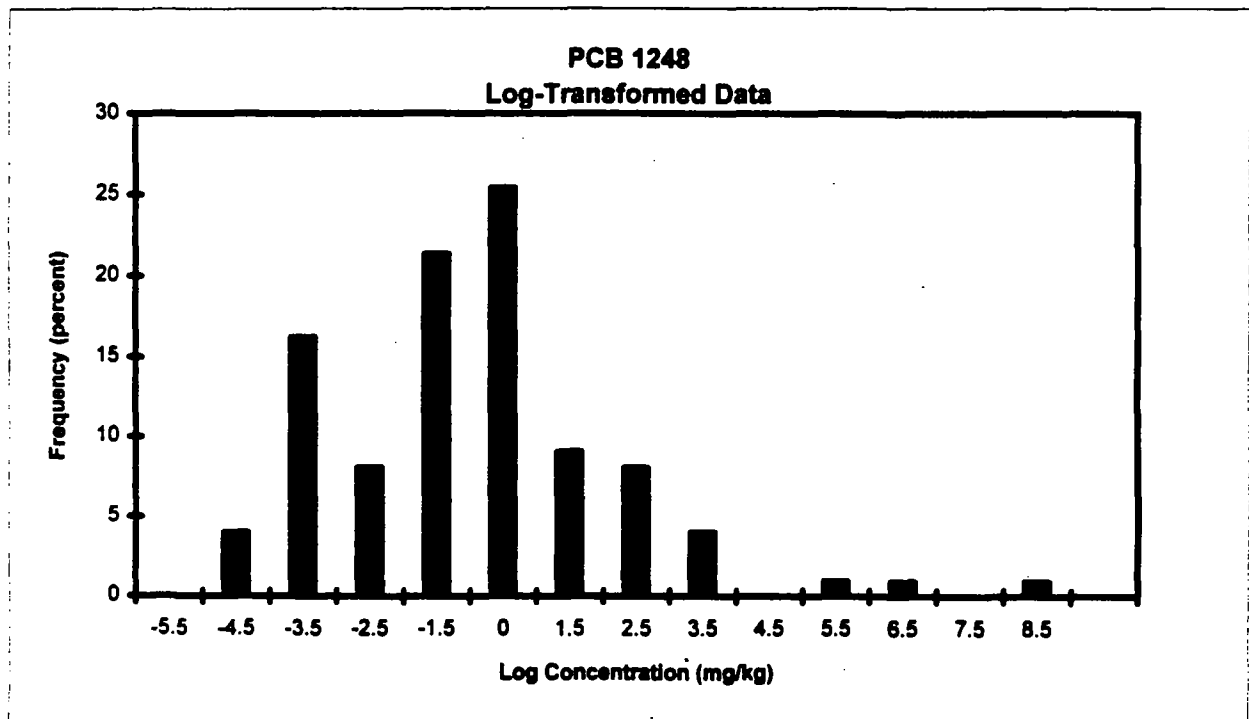
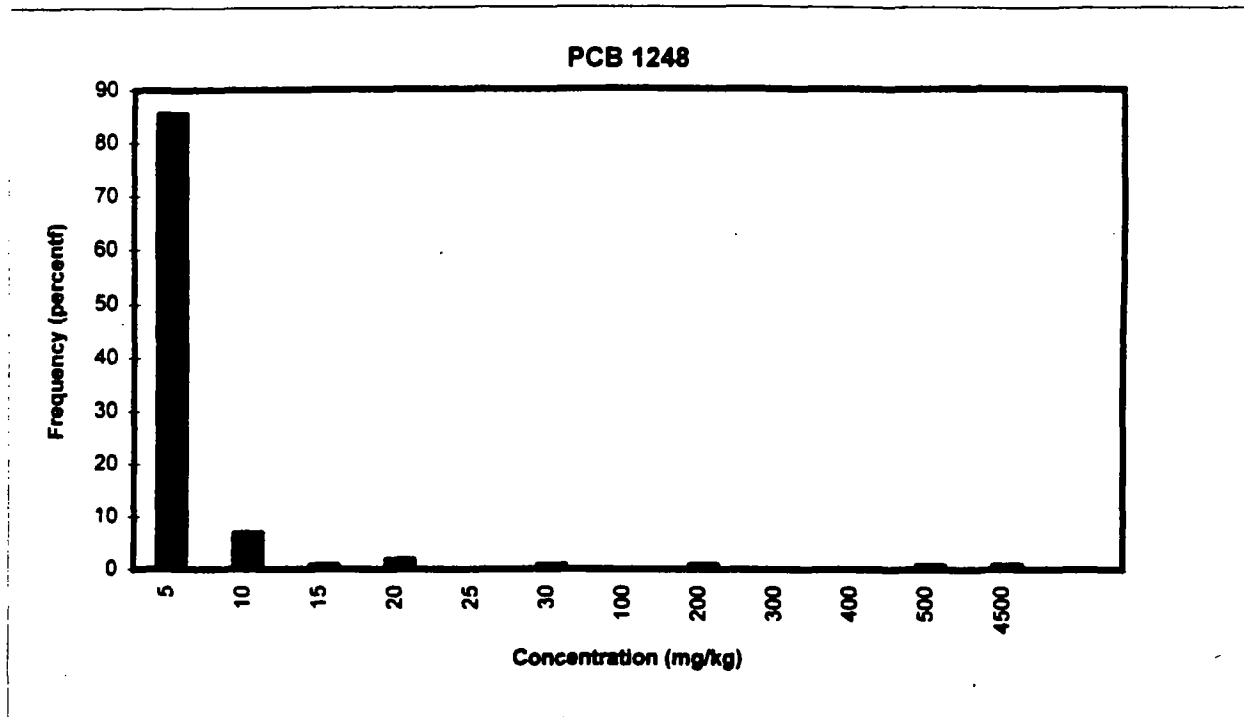


Figure A5-3
Production Area Combined Surface and Subsurface Soils
PCB 1248 Data Distribution

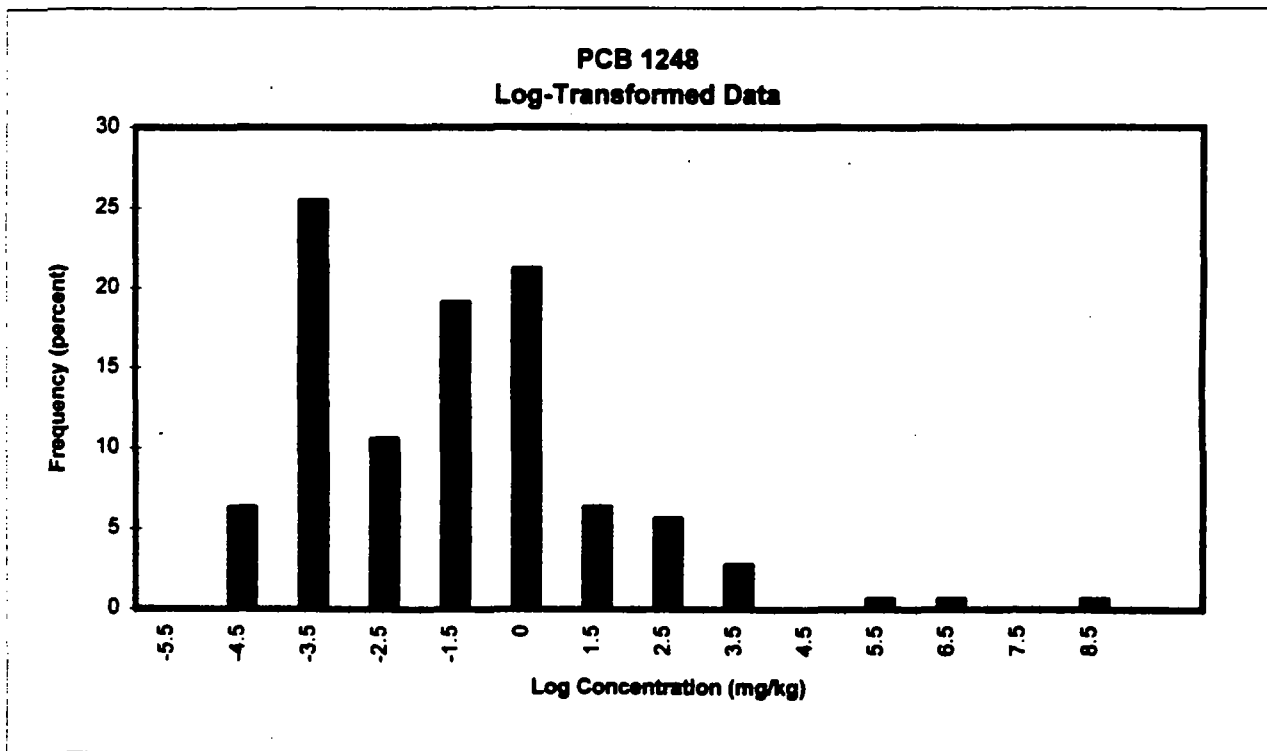
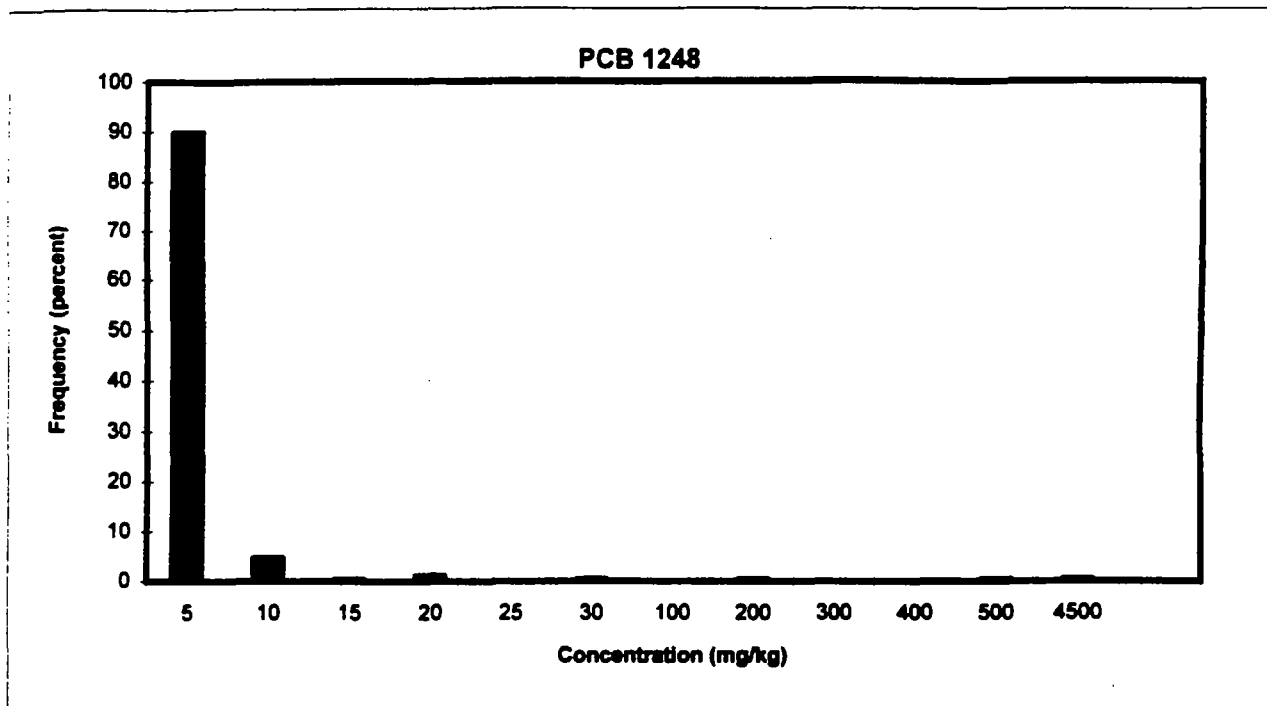


Figure A5-4
Production Area Surface Soil
PCB 1254 Data Distribution

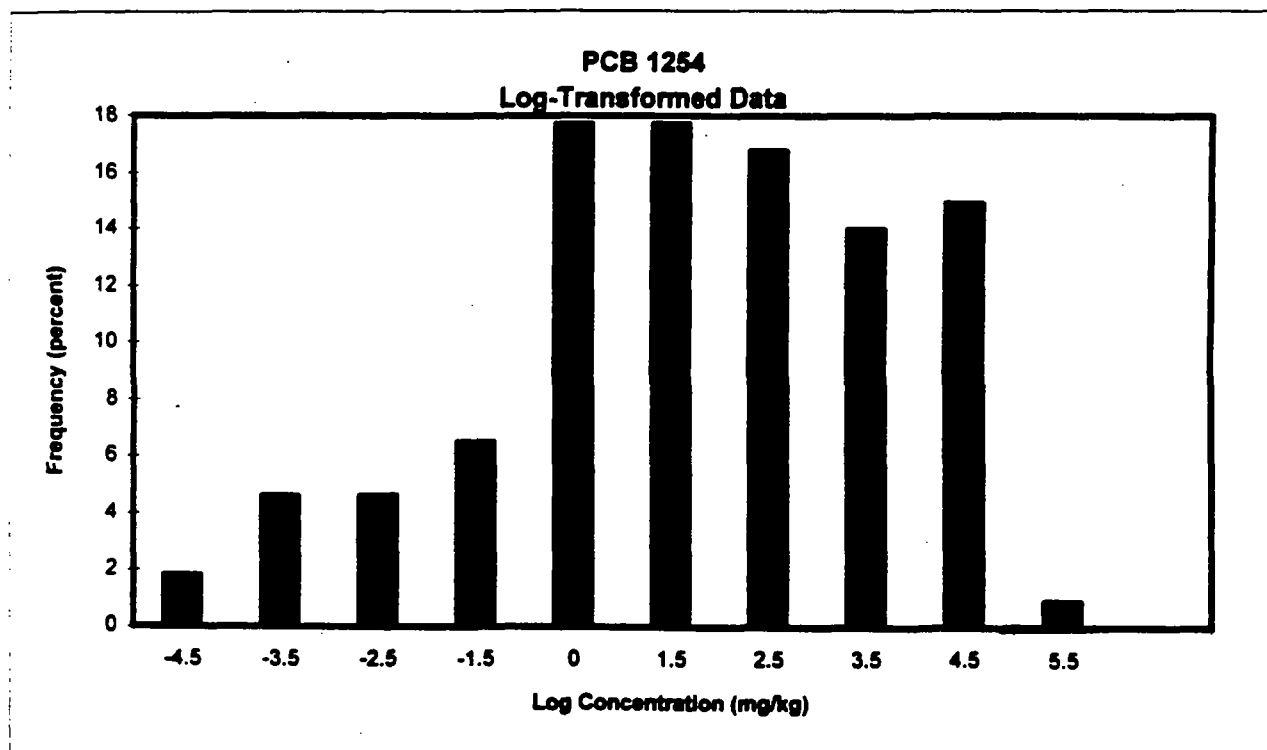
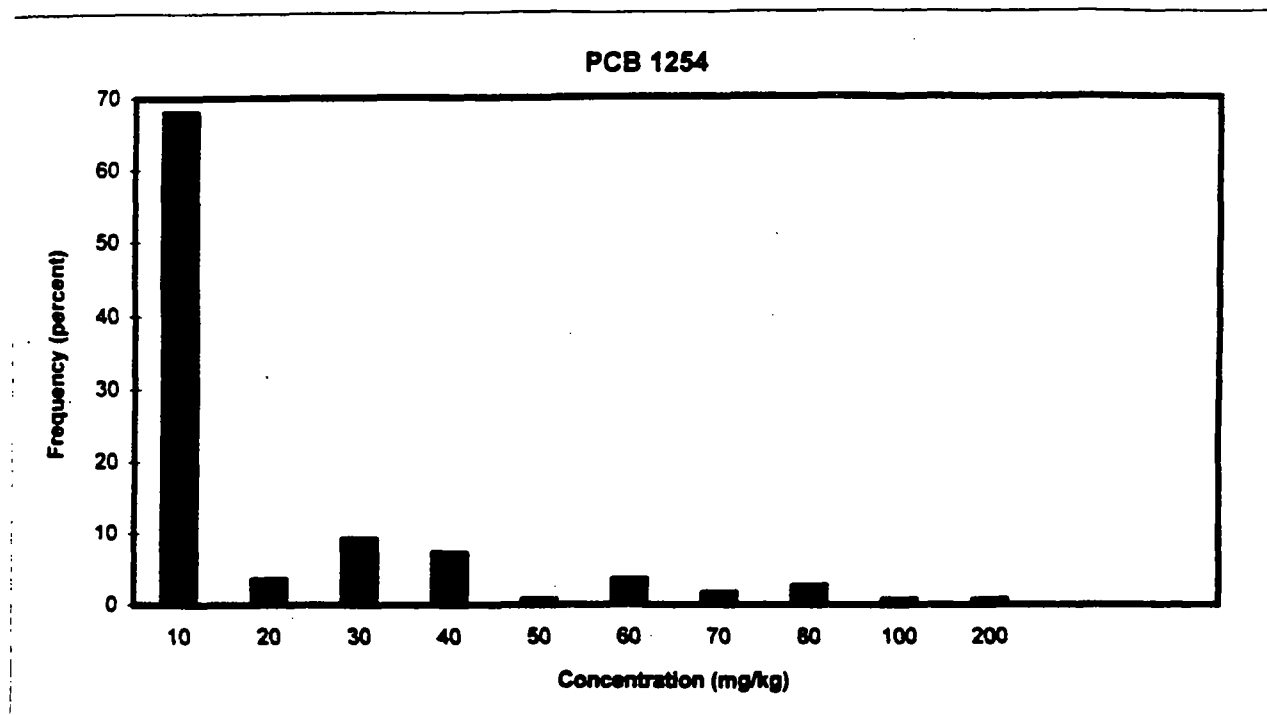
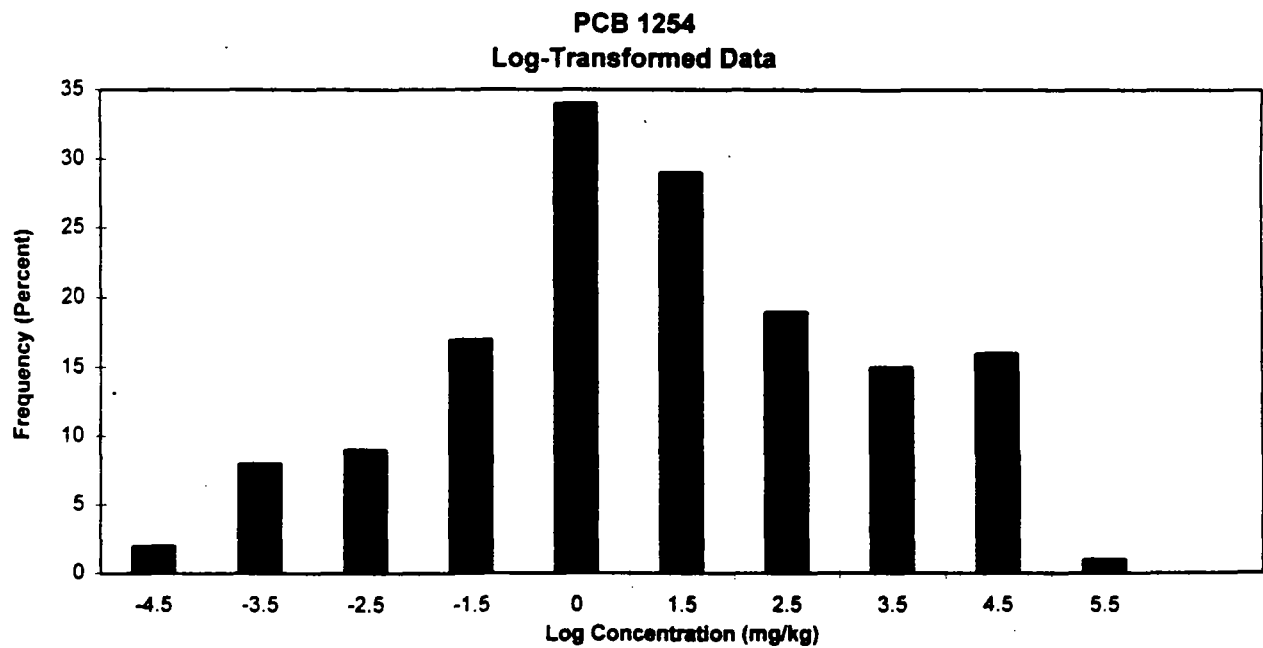
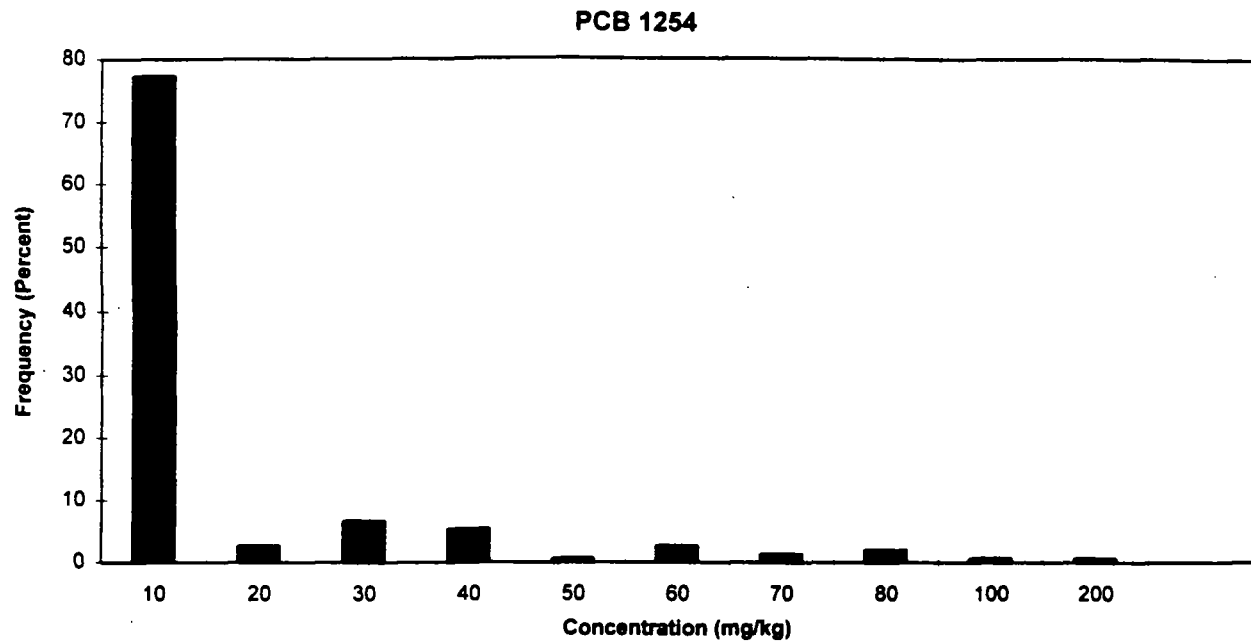
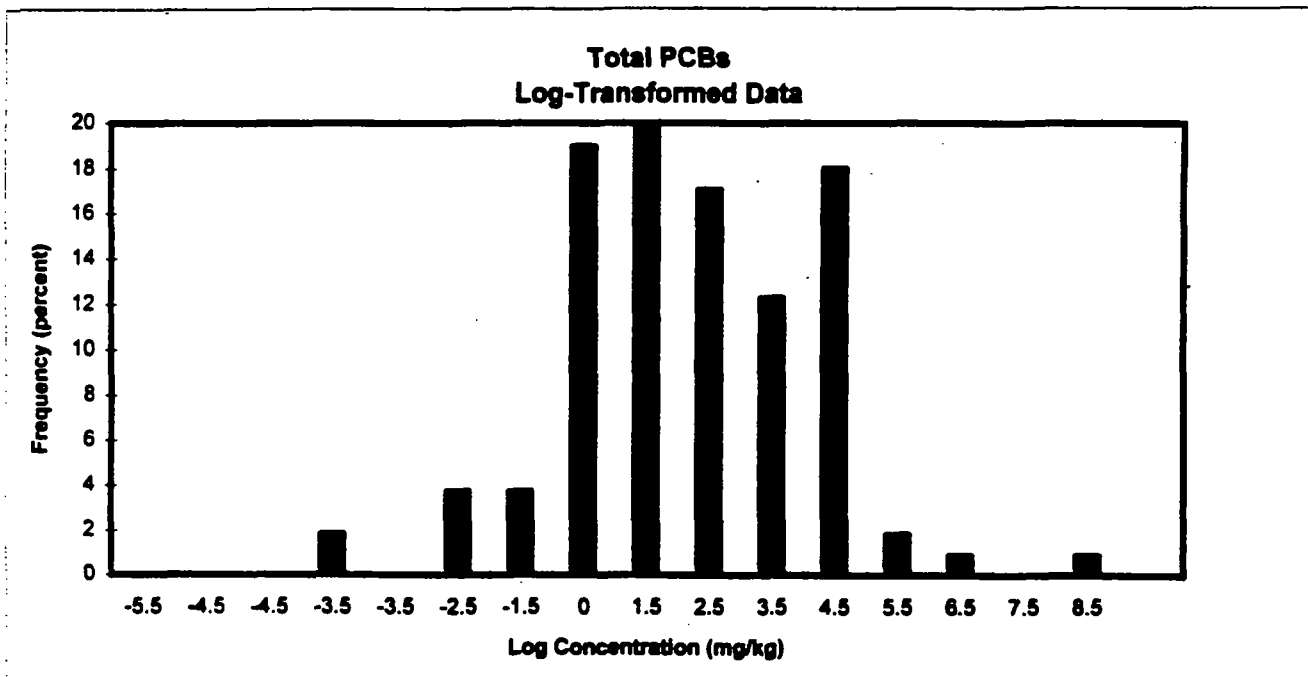
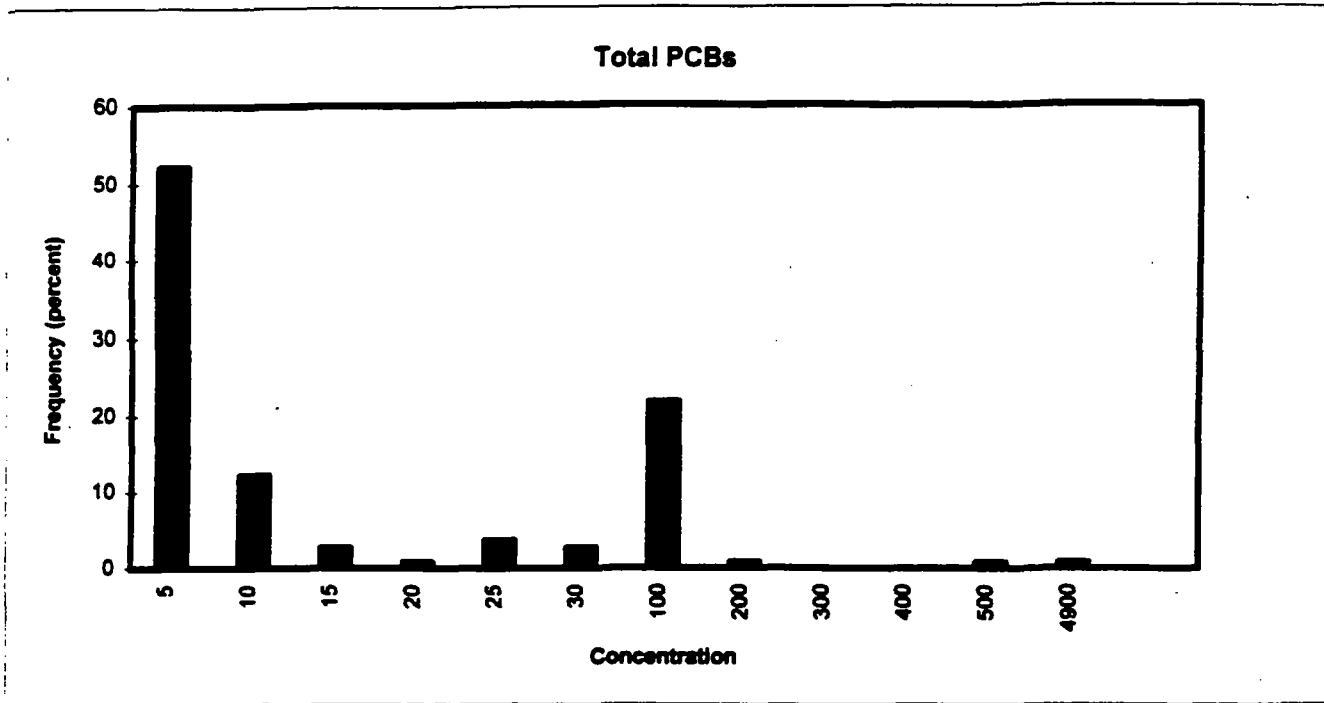


Figure A5-5
Production Area Combined Surface and Subsurface Soil
PCB 1254 Data Distribution



**Figure A5-6
Production Area Surface Soil
Total PCBs Distribution**



Section A6

Toxicity Assessment

A6.0 Toxicity Assessment

Toxicity assessment consists of identifying and evaluating toxicity criteria and health effects information for the chemicals detected in impacted and/or potentially impacted media. In the Risk Assessment, toxicity criteria were identified during the COPC screening process (Attachment 2). Attention is given to the relationship between the level of the exposure and the severity of any resultant adverse health effects. Specific adverse health effects are noted for each chemical carried through the risk assessment process, particularly those effects on which the toxicity criteria are based. Information obtained during the toxicity assessment is used in the risk characterization (Section A7.0) to estimate risks associated with the exposure levels estimated during the exposure assessment (Section A5.0).

Toxicity information for the COPC is shown in Table A6-1 and the full Integrated Risk Information System (IRIS) print-outs are given in Attachment 5. This information includes the following:

- Chronic reference doses (RfDs);
- Cancer slope factors (CSFs);
- Target organs for adverse health effects;
- Tumor sites; and
- USEPA weight-of-evidence classification system for cancer effects.

The items listed above are described in the following subsections.

A6.1 Health Effects Classification

Chemicals may exhibit a variety of adverse health effects. For risk assessment purposes, these adverse effects are generally divided into two categories: noncancer and cancer. The reason for this distinction is the opinion that the mechanism for each is different. It is generally believed that the body has protective mechanisms against most noncancer effects. These defenses must be overcome by a given exposure level of a toxicant before any adverse effects occur. Therefore, it is thought that a range of exposure levels from zero to some finite threshold level can be tolerated with essentially no risk of adverse health effects.

Unlike noncancer effects, cancer is assumed by USEPA not to have a threshold level (USEPA, 1989a). The hypothesized mechanism of carcinogenesis assumes that there is essentially no

level of exposure to a carcinogen that does not pose a finite probability, however small, of generating a carcinogenic response.

The USEPA-preferred and most regularly updated source of toxicity information is the (IRIS) on-line data base. IRIS was the primary source of health effects criteria used in this toxicity assessment, and IRIS toxicity profiles are included as Attachment 5. When health effects criteria were not found in IRIS, this information was sought in the Health Effects Assessment Summary Tables (HEAST-USEPA, 1994), the agency's second preference. Other sources of toxicity information were used only when the health effects criteria were not available in IRIS or HEAST.

Health effects criteria for noncancer effects and cancer effects are discussed in Sections A6.2 and A6.3, respectively. A given chemical may exhibit both noncancer and cancer effects.

A6.2 Health Criteria for Noncancer Effects

The assessment of toxic effects for a noncarcinogenic chemical is based on the RfD. An RfD is a daily human intake level measured in milligrams of chemical per kilogram of body weight (mg/kg-day), based on the oral ingestion pathway and developed or verified by USEPA's RfD/RfC Work Group. RfD values are derived from toxicity data to be within a tolerable threshold level, such that a lifetime of exposure to a given toxicant at the RfD level theoretically poses virtually no risk of deleterious effects (USEPA, 1989a). Reference concentrations (RfCs) are developed or verified for inhalation also by USEPA's RfD/RfC Work Group. An RfC is based on a constant lifetime average concentration of a chemical in air, measured in milligrams of chemical per cubic meter of air (mg/m³). Likewise, they are derived from toxicity data to be within a tolerable threshold level that poses virtually no risk of deleterious health effects.

RfCs may be converted to provisional inhalation route RfDs using exposure assessment calculations. Note that in Table A6-1, provisional inhalation route RfDs are referred to as "RfD_is", and oral route RfDs are referred to as "RfD_os".

RfD_os are also used for the dermal absorption route of exposure. Chemical-specific differences of absorption via the oral and dermal routes are addressed separately in the exposure assessment (Section A5.0 and Attachment 3). Even though RfDs are derived to be below threshold health effects levels using conservative assumptions, it cannot be definitively stated that a given level

of exposure below the RfD poses no risk. Neither can it be assumed that a given exposure level above the RfD poses a definite human health risk. The most sensitive subpopulations are considered in establishing RfDs.

An RfD is derived from human studies that provide some quantification of exposure or animal studies. If available, a no-observed-adverse-effects level (NOAEL) is used. Uncertainty factors, typically of an order of magnitude each, may be used to account for the following:

- Variations in sensitivity among the exposed population;
- Extrapolations from animal studies to human exposures;
- Extrapolations from shorter term studies to chronic exposures; and
- Extrapolations from a lowest-observed-adverse-effects level (LOAEL) to a NOAEL.

An additional uncertainty or modifying factor is used to reflect professional judgement of the uncertainties of the study and the database not explicitly addressed by the above factors. The modifying factor may range from one to less than ten. When combined, these uncertainty factors may result in a nearly 10,000-fold margin of safety with respect to the toxicity criteria. Therefore, an RfD or RfC is biased in overestimating the possibility of toxic effects from exposure to a chemical.

A6.2.1 RfD for PCB 1248

PCB 1248 has no USEPA-established reference dose (RfD), so it is necessary to derive a provisional RfD. PCB 1248 elicits both developmental and immunologic effects, with developmental appearing to be the critical effect. A provisional PCB 1248 RfD of 8×10^{-5} mg/kg-day was derived for developmental effects (Table A6-1), and a provisional RfD of 1×10^{-3} mg/kg-day for immunologic effects was also derived. Because of potential additive toxicity with PCB 1254, immunologic effects of PCB 1248 are relevant to the Risk Assessment. A detailed discussion of how these provisional RfDs were developed is given in the following subsections.

A6.2.1.1 Developmental Effects

In addition to IRIS, the Hazardous Substances Data Bank (HSDB), Registry of Toxic Effects of Chemical Substances (RTECS), Developmental and Reproductive Toxicology Database (DART), and TOXLINE on-line databases were searched for toxicity information on PCB 1248.

The journal articles referenced in these databases were reviewed. As mentioned above, the critical effect that occurs at the lowest dose is a developmental effect. In the key study (Allen et al., 1979) adult female rhesus monkeys (eight per exposure level) were fed PCB 1248 at estimated doses of 0.008 and 0.016 mg/kg-day for 18 months. After seven months of exposure, the primates were bred and the mothers and offspring evaluated for toxic effects. Six of eight conceptions at the lower exposure level, and seven of eight at the higher level resulted in live births, and the infants survived the experimental period. No maternal toxicity was observed at either dose level, but the infants were somewhat smaller than controls at birth. These infants gained less weight than controls during the nursing period, and they developed focal areas of skin hyperpigmentation. These are some of the classic signs of PCB intoxication. A PCB 1248 RfD of 8×10^{-5} mg/kg-day for people is estimated using the following uncertainty factors:

- Extrapolation from a lowest-observed adverse effect level to a NOAEL = 10
 - The standard default value was used because several of the 16 female rhesus monkeys in the combined 0.008 and 0.016 mg/kg-day dose groups (all of which conceived) had resorptions/abortions (Allen et al., 1979). Unfortunately, the reproductive performance of the control group is not specified. Reproductive performance in rhesus monkeys is highly variable among colonies, but 25 to 35% fetal losses in pregnant females is common. However, other publications by this group of investigators report no fetal losses in control groups for PCB studies conducted in the same time frame as that of Allen et al. (1979). Even though this level of reproductive performance is highly unusual, it can only be assumed from the information given that no fetal losses were experienced in the control group. Otherwise, a LOAEL to NOAEL uncertainty factor of 3 could be justified. The somewhat lower birth weights and weight gain observed in the study relative to controls was not characterized by the authors as statistically significant. Schantz et al. (1989) made similar observations in PCB 1248 rhesus monkey studies conducted at the same laboratory at maternal exposure levels of 0.016 and 0.040 mg/kg-day. They also characterized the hyperpigmentation of infants as mild, and reversible after weaning at these exposure levels. This implies that the 0.008 mg/kg-day exposure level in the Allen et al. (1979) study is close to a NOAEL dose. This justification is similar to that used by the USEPA for using the uncertainty factor of 3 for NOAEL estimation in deriving the RfD for PCB 1254 because of the less severe effects on periocular tissues and nail beds in rhesus monkeys at lower doses (IRIS, 1995; see Attachment 5).

- Extrapolation from rhesus monkeys to man = 1
 - Explanation: The vast majority of differences in the severity of toxic effects at similar dose levels of a given chemical among test animal species is related to differences in metabolism and toxicokinetics. Comparative PCB metabolism and toxicokinetic studies in man relative to monkeys, dogs, and rats show that these species handle PCBs in a manner similar to people (Schnellman et al., 1983, 1984, 1985). Monkeys match best with the human data, a conclusion which is corroborated by the Agency for Toxic Substances Disease Registry (ATSDR, 1991) and the USEPA (IRIS, 1995; see Attachment 5, page 15). This close similarity between monkeys and humans based on data that are rarely available in people, and the fact that rhesus monkeys exhibit adverse PCB health effects at doses ten-fold lower than in other species, justifies direct extrapolation to people.
- Human variability = 10
 - Explanation: Standard default.

A6.2.1.2 Immunologic Effects and Potential Additive Toxicity

The potential for additive toxicity of PCB 1248 and PCB 1254 was evaluated. According to USEPA guidance, additivity is to be considered if two or more compounds affect the same target organ or have the same mechanism of action (USEPA, 1989a). Developmental toxicity, the critical effect of PCB 1248, is not listed in IRIS or any other database searched as a critical effect of PCB 1254. Immunological effects are a critical effect listed in IRIS for PCB 1254. PCB 1248 also elicits immunologic effects. Therefore, potential additive effects of PCB 1248 and PCB 1254 were evaluated with respect to immunologic effects. The application of additive toxicity is discussed in the risk characterization (Section A7.0).

The lowest dose at which an immunologic effect was observed for PCB 1248 is 0.2 mg/kg-day (Thomas and Hinsdill, 1978). After eleven months on experimental diets resulting in a dose level of either 0.1 or 0.2 mg/kg-day, two groups of eight rhesus monkeys were injected intravenously with sheep erythrocytes (SRBCs). A third, control group was likewise injected with SRBCs. Compared to the 0.1 mg/kg-day and control groups, the 0.2 mg/kg-day group showed a significantly reduced SRBC antibody titer one week after primary immunization. At a dose of 0.1 mg/kg-day, no immunologic effect was observed. This lower dose is regarded as a NOAEL for immunologic effects. An uncertainty factor of 10 to extrapolate chronic exposure,

a factor of 10 to account for human variability, and a factor of 1 to extrapolate from rhesus monkeys to humans (Section A6.2.1.1) were used to estimate a PCB 1248 provisional RfD for immunologic effects. If the NOAEL for immunologic effects (0.1 mg/kg-day) is divided by the combined factor of 100, the resulting provisional RfD is 1×10^{-3} mg/kg-day. This value is about 12 times greater than the provisional RfD calculated for developmental effects.

As shown in Table A6-1, the established RfD for PCB 1254 is 2×10^{-5} mg/kg-day. This value is based on ocular exudate, meibomian gland effects, distorted growth of nails, and decreased antibody response to SRBCs in rhesus monkeys dosed at 5×10^{-3} mg/kg-day (IRIS, 1995). The provisional RfD of PCB 1248 with regard to immunologic effects is 50 times higher than the RfD for PCB 1254.

Even though the critical effects of PCB 1248 and PCB 1254 are different, potential additive immunologic effects may affect the estimation of MPS values. As stated above, the RfD for PCB 1254 is 50 times lower than the provisional RfD for PCB 1248 based on immunologic effects. Therefore, 50 mg/kg of PCB 1248 equals 1 mg/kg of "PCB 1254 equivalents" in the use of this relationship to estimate acceptable residual PCB soil concentrations.

Additivity with regard to developmental effects might also be pertinent if PCB 1248 was detected at significantly higher concentrations than PCB 1254 at the Site. However, in the databases that exist for site soil, PCB 1254 is detected with greater frequency and generally at higher concentrations than PCB 1248. Thus, the critical immunologic effects of PCB 1254 and the additive immunologic effects of PCB 1248, from a toxicity viewpoint, "drive" the estimation of MPS values for PCBs.

A6.3 Health Criteria for Cancer Effects

Human carcinogens and potential human carcinogens are categorized into the following groups by USEPA Human Health Assessment Group's weight-of-evidence classification system:

- **Group A**
Human Carcinogen (sufficient evidence of carcinogenicity in humans).
- **Group B**
Probable Human Carcinogen (B1--limited evidence of carcinogenicity in humans; B2--

sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans).

- **Group C**

Possible Human Carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of human data).

- **Group D**

Not Classifiable as to Human Carcinogenicity (inadequate or no evidence).

- **Group E**

Evidence of Noncarcinogenicity for Humans (no evidence of carcinogenicity in adequate studies).

Quantitative cancer risk assessments are performed on chemicals in Groups A and B, and on a case-by-case basis for Group C. The quantification of potential human cancer risks exhibited by a chemical is based on its cancer slope factor (CSF). In practical terms, a CSF is an estimate of the risk associated with a chronic daily intake of one milligram of chemical per kilogram of body weight (mg/kg-day)⁻¹. Separate CSFs are derived for the oral (CSF_o) and inhalation (CSF_i) exposure routes. Typically, IRIS lists no CSF_i value, but instead lists an inhalation unit risk (UR_i). The UR_i is the potential cancer risk associated with an average lifetime exposure to an airborne concentration of one microgram of a chemical per cubic meter of air (μg/m³)⁻¹. UR_i values can be converted to provisional CSF_i values using exposure assessment methodologies. Similar to the case of noncancer effects (Section A6.2), CSF_o values may be used for the dermal absorption exposure route, using chemical-specific factors to adjust for the differences in absorption between the oral and dermal routes.

CSFs are calculated through the use of mathematical extrapolation models. Generally, the USEPA limits its extrapolation to the linearized, multistage model, despite heavy criticism from the scientific community. This model incorporates data from studies performed using a relatively high dose, and estimates the largest possible linear slope within the 95th percentile upper confidence limit, extrapolating the study data to a low dose. Because of the choice of mathematical model and of the 95th percentile upper confidence limit, the CSF represents a conservative upper-bound estimate of the true cancer risk of a chemical to humans.

Table A6-1
Toxicity Summary for Compounds of Potential Concern

Chemical	RfD _o ^a Oral (mg/kg-day)	RfD _i ^b Inhalation (mg/kg-day)	Target Organ ^c	Cancer WOE ^d	CSF _o ^e Oral (mg/kg-day)	CSF _i ^f Inhalation (mg/kg-day)	Tumor Site(s) ^g
PCB 1248	8×10^{-5} ^h	(see RfD _o) ⁱ	Developmental effects				
PCB 1254	2×10^{-5}	(see RfD _o) ⁱ	Decreased antibody response; eyes; nail beds				
PCB 1260				B2	7.7×10^0	(see CSF _o) ^j	Liver (hepatocellular carcinoma; neoplastic liver nodules)
<i>gamma</i> -Chlordane	6×10^{-5}		Liver	B2	1.3×10^0	1.3×10^0	Liver (hepatocellular carcinomas)
2-Nitroaniline	(see RfD _i) ^k	5.71×10^{-5} ^l	Blood				
Methoxychlor	5×10^{-3}	(see RfD _o) ⁱ	Loss of litters in (rabbits)	D			
Aldrin	3×10^{-5}		Liver	B2	1.7×10^1	1.7×10^1	Liver carcinoma
Beryllium	5×10^{-3}		(None)	B2	4.3×10^0 ^m	8.4×10^0 ⁿ	Lung cancer, osteosarcomas
Dieldrin	5×10^{-5}		Liver	B2	1.6×10^1	1.6×10^1	Liver carcinoma
Heptachlor epoxide	1.3×10^{-5}		Decreased liver weight	B2	9.1×10^0	9.1×10^0	Liver carcinoma

^aChronic reference dose, oral route. Source: Integrated Risk Information System database (IRIS), unless otherwise noted.

^bChronic reference dose, inhalation exposure route. Calculated from reference concentrations (RfCs).

^cSource: Same as for the RfD value(s).

^dUSEPA weight-of-evidence (WOE) classification system regarding carcinogenic effects. Source: IRIS, unless otherwise noted.

^eCancer slope factor, oral exposure route. Source: IRIS, unless otherwise noted.

^fCancer slope factor, inhalation route. Source: IRIS, unless otherwise noted.

^gSource: Same as for the CSF value.

^hNo toxicity data available in IRIS or the *Health Effects Assessment Summary Tables* (HEAST) (USEPA, 1994). Values were derived from a rhesus monkey toxicity study (Allen et al., 1979). The monkeys were given a dose equivalent to 8×10^{-3} mg/kg-day for approximately 1.5 years. This dose was divided by an uncertainty factor of 100. This includes a factor of 10 to account for human variability and a factor of 10 to extrapolate from a lowest-observed-adverse-effects level to a no-observed-adverse-effects level. A provisional PCB 1248 RfD of 1×10^{-3} mg/kg-day was estimated for immunologic effects, the critical effect of PCB 1254. Refer to Section A6.2 for discussion of the immunologic effects RfD for PCB 1248 and potential additive toxicity of PCB 1248 and PCB 1254.

ⁱNo RfD_i available in IRIS or HEAST; RfD_o value was substituted in the Risk Assessment.

^jNo CSF_i available in IRIS or HEAST; CSF_o value was substituted in the Risk Assessment.

^kNo RfD_o available in IRIS or HEAST; RfD_i value was substituted in the Risk Assessment

^lDerived from the RfC of 2×10^{-4} (mg/m³). HEAST (USEPA, 1994).

^mNo CSF_o available in IRIS or HEAST; value shown was estimated from the inhalation unit risk (UR_i) of 2.4×10^{-3} (ug/m³)⁻¹. Source of UR_i: IRIS.

ⁿValue was estimated from the UR_i value of 2.4×10^{-3} (ug/m³)⁻¹. Source of UR_i: IRIS.

Section A7

Risk Characterization

A7.0 Risk Characterization

The objective of risk characterization is to evaluate and quantify the potential risks associated with a site. This is done by combining the exposure levels estimated in the exposure assessment (Section A5.0) with the appropriate toxicity criteria identified during the toxicity assessment (Section A6.0) to quantitatively estimate potential cancer risk for carcinogens and the potential for noncancer adverse health effects. Because of basic differences in the mechanisms of toxicity, the risks associated with cancer and noncancer adverse health effects of chemicals are characterized separately. Risk characterization methodologies used in the Risk Assessment are consistent with the HHEM and are described in Attachment 6. The following provides an overview of the process used in risk characterization.

The total hazard index (THI) represents the overall calculated noncancer risks posed by the COPC in a given exposure scenario. The calculation of the THI and associated values such as hazard quotients (HQs) and hazard indices (HIs) are described in detail in Attachment 6. Briefly the THI is the sum of the separate chemical-specific HQ values for all of the COPC, via all the relevant routes of exposure for the exposure scenario. The HQ is calculated by dividing the estimated chemical intake level (IN) to a chemical, via one exposure pathway, by the appropriate RfD. Both the IN and the RfD are given in units of milligrams of chemical per kilogram of body weight per day (mg/kg-day). Thus, if the IN is greater than the RfD, the HQ will exceed a threshold value of 1. The chemical-specific HI is the sum of all HQ values (via all exposure pathways) for a particular COPC.

To evaluate noncancer risk, the THI is compared to a target value of 1. The THI is rounded to one significant figure in accordance with the HHEM. If the THI is less than or equal to 1, then it is unlikely, given the exposure assumptions, that the COPC present a health risk. If the THI (rounded to one significant figure) exceeds 1, then separate THI values should be calculated for the separate target organs. If any of the resultant target organ-specific THI values exceed the target value of 1, then a potential for adverse health effects may be indicated. When exposure to multiple chemicals with the same target organ exist, the combined effect of the chemicals may be additive, synergistic, antagonistic, or they may have no influence on one another at all. Antagonistic relationships result in health effects that are less than those predicted by a chemical given alone; synergistic relationships result in health effects that exceed the results predicted by a chemical given alone and the additive effects of chemicals with similar effects. Combined noncancer health effects on the same target organ are assumed to be additive in this Risk Assessment. It should be noted that the THI value is to be compared to the threshold value of 1,

and should not be used as an independent, quantitative estimator of risk. The reasons for this are related to the assumption discussed in Section A6.2 of the toxicity assessment that a threshold level of exposure must be exceeded before chemicals elicit adverse noncancer health effects.

The total incremental lifetime cancer risk (ILCR) is the sum of all estimated potential cancer risks associated with all carcinogenic chemicals in a given exposure scenario. Combined cancer risks associated with exposure to multiple carcinogens are assumed to be additive, unless available information suggests otherwise. In weighing exposures to potentially carcinogenic compounds, a reasonable level of risk must be selected. Cancer is of significant occurrence in the United States with an estimated lifetime risk of developing cancer being about three out of every ten people (3×10^{-1}) (American Cancer Society, 1990). Approximately 80 percent of these cases result in death directly attributable to the disease. The USEPA regards an ILCR of between 1×10^{-6} (1 in 1,000,000) and 1×10^{-4} (1 in 10,000) as acceptable. Thus, this may be interpreted as an increase in the United States baseline cancer incidence from 300,000 per million population to a range of 300,001 to 300,100 per million population. Under the Resource Conservation and Recovery Act (RCRA), this is regarded as the protective risk range for media protection standards (USEPA, 1990b). Alternatively, a project-specific target risk range or risk level may be used. If the ILCR exceeds the upper bound of the target risk range, then further evaluation or corrective action may be indicated.

A7.1 Special Considerations of PCBs

A7.1.1 PCB 1248 and PCB 1254

Section A6.2.1 discusses the differences in the respective critical effects for PCB 1248 and PCB 1254. PCB 1254 has an RfD of 2×10^{-5} mg/kg-day based on immunologic effects. A provisional RfD of 8×10^{-5} mg/kg-day was derived for PCB 1248, based on developmental effects. This developmental effects RfD was used in the risk characterization. Because the RfD for PCB 1254 and the provisional RfD for the critical effect of PCB 1248 are based on different target organs and mechanisms of toxicity, hazard indices that result from these RfDs are not additive.

A provisional RfD was also derived for PCB 1248 that is specific for immunologic effects; this

value is 1×10^{-3} mg/kg-day. This immunologic-based RfD for PCB 1248 was also used in the risk characterization. The resultant HI is summed with the HI for PCB 1254 to estimate an additive effects THI for immunologic effects, referred to as the "Combined PCB THI".

A7.1.2 Total PCBs

The analytical results of all detected PCBs were summed and referred to as total PCBs. These are PCB 1248, PCB 1254, and PCB 1260 in the Production Area; and PCB 1248 and PCB 1254 in the Warwick Area. The resultant data set was treated as if total PCBs were a different chemical. Total PCBs was used in the risk characterization, using the Region I policy assumption that the combination of all PCBs is equal in cancer potency to PCB 1260 (USEPA 1995b). This practice is not consistent with PCBs toxicity data. A large toxicity database exists for PCB 1254, from which it is concluded that it is not carcinogenic. Also, existing studies suggest that PCB 1248 is not carcinogenic. Since most of the PCBs detected at the Site are PCB 1254 and PCB 1248, to assume that these mixtures are carcinogens with the same cancer potency as PCB 1260 grossly overestimates potential cancer risks.

A7.2 Risk Characterization Results

A7.2.1 Production Area

Production Area noncancer and cancer effects risk characterization results are summarized in Table A7-1.

The Combined PCBs THI for the Production Area on-site worker is estimated as 0.07. The HI for the developmental effects of PCB 1248 is 0.002. These values are less than the target value of 1. Thus, adverse noncancer health effects associated with Site soils are unlikely to occur in the Production Area. Regarding potential cancer risks, the total ILCR is estimated as 6×10^{-6} , with PCB 1260 accounting for over 99% of the estimated potential total ILCR. This is within the RCRA protective risk range of 10^{-6} to 10^{-4} .

Overall, both cancer and noncancer potential human health risk estimates are below their respective "action" criteria for the on-site worker in the Production Area. Region I policy is to assume that all PCBs have a cancer potency equal to that of PCB 1260. The total PCBs data set was found to be lognormally distributed (Section A2.0), whereas the data set for PCB 1260 was assumed, due to a low number of detections, to be normally distributed. Thus, the exposure point concentration used for the total PCBs data set is the 95th percentile UCL of the geometric

mean. This value (5.9 mg/kg) is less than the 95th percentile UCL of the arithmetic mean (6.1 mg/kg) used as the exposure point concentration of PCB 1260. Therefore, to be conservative Table A7-1 depicts the potential cancer risk estimated for total PCBs to equal that of PCB 1260. As stated above, this value of 6×10^{-6} is within the RCRA protective cancer risk range.

A7.2.2 Warwick Area

The THI for the hypothetical Warwick Area on-site resident is estimated as 0.6 (Table A7-2). This is estimated by combining the HI values of PCB 1254 and PCB 1248, and assuming that these effects are additive. This value is less than the target THI criterion value of 1. The HI value of methoxychlor is 0.14, and its critical effect is listed in IRIS as decreased litter sizes in rabbits. It is appropriate to combine this value with the developmental HI for PCB 1248 (0.42). The resultant THI, rounded to one significant figure, is also a value of 0.6. 2-Nitroaniline has an HI value of 0.34. Because its critical effects are neither immunologic nor developmental, it is inappropriate to assume additivity of 2-nitroaniline with the other COPC.

The total ILCR for the hypothetical future on-site resident is estimated as 1×10^{-4} . This value is within the RCRA protective risk range of 10^{-6} to 10^{-4} . However, the USEPA Region I policy is to assume that all PCBs have a cancer potency equal to that of PCB 1260 (USEPA, 1995b). Total PCBs for the Warwick Area is comprised of only PCB 1248 and PCB 1254. As stated in (Section A7.2.1), this practice is not consistent with PCB toxicity data. This is particularly true for the Warwick Area where no PCB 1260 was detected. To assume that these compounds have the same cancer potency as PCB 1260, when they are regarded as noncarcinogenic, greatly exaggerates potential cancer risks. The ILCR of the COPC excluding total PCBs is 2×10^{-5} . This value also is within the RCRA protective risk range (10^{-6} to 10^{-4}).

Table A7-1
Risk Summary for Production Area
On-Site Worker Scenario

Noncancer Risks

CHEMICAL	HAZARD QUOTIENT			HAZARD INDEX
	Ingestion	Dermal	Inhalation	
PCB 1248-Dev. ^a	0.00086	0.0013	0.000016	0.0022
PCB 1248-Imm ^b	0.000069	0.00010	0.0000013	0.00022
PCB 1254	0.028	0.042	0.00017	0.070
TOTAL HAZARD INDEX ^c				0.07

Cancer Risks

CHEMICAL	CANCER RISK			
	Ingestion	Dermal	Inhalation	Combined Routes
PCB 1260	2.5×10^{-6}	3.7×10^{-6}	1.7×10^{-8}	6.1×10^{-6}
<i>gamma</i> -Chlordane	8.8×10^{-9}	2.6×10^{-8}	3.1×10^{-10}	3.5×10^{-8}
Total PCBs ^d	2.5×10^{-6}	3.7×10^{-6}	1.7×10^{-8}	6.1×10^{-6}
TOTAL LIFETIME INCREMENTAL CANCER RISK ^e				6×10^{-6}

^aNoncancer risks based on developmental effects.

^bNoncancer risks based on immunologic effects.

^cAssumes additivity for the effects of PCB 1254, and the immunologic effects of PCB 1248.

^dIn accordance with USEPA Region I policy, risk of total PCBs was estimated assuming that all PCBs have the same cancer potency as PCB 1260. This policy contradicts toxicological data which indicate that PCB 1248 and PCB 1254 are noncarcinogenic. The total PCBs data set was created from the combined concentrations of PCB 1248, PCB 1254, and PCB 1260 in surface soil (refer to Section A5.3.1). The total PCB data set was found to be lognormal; due to a paucity of detects, the PCB 1260 data set was assumed to be normal. The 95th percentile upper confidence limit (UCL) of the (geometric) mean concentration for the total PCBs data set (5.9 mg/kg) is less than the UCL of the (arithmetic) mean concentration for PCB 1260 (6.1 mg/kg). For conservativeness, the UCL of the PCB 1260 data set was used for total PCBs.

^eIncludes the cancer risks associated with *gamma*-Chlordane and PCB 1260.

Table A7-2
Risk Summary for Warwick Area
On-Site Residential Scenario

Noncancer Risks

CHEMICAL	HAZARD QUOTIENT			HAZARD INDEX
	Ingestion	Dermal	Inhalation	
PCB 1248-Dev. ^a	0.32	0.11	0.0032	0.42
PCB 1248-Imm ^b	0.025	0.008	0.00025	0.034
PCB 1254	0.44	0.15	0.0012	0.58
2-Nitroaniline	0.21	0.13	0.0044	0.34
Methoxychlor	0.078	0.051	0.000027	0.13
TOTAL HAZARD INDEX ^c				0.6

Cancer Risks

CHEMICAL	CANCER RISK			
	Ingestion	Dermal	Inhalation	Combined Routes
Aldrin	2.4×10^{-6}	1.6×10^{-6}	4.3×10^{-8}	4.0×10^{-6}
Beryllium	2.1×10^{-6}	1.2×10^{-5}	1.4×10^{-9}	1.4×10^{-5}
Dieldrin	1.7×10^{-6}	1.1×10^{-6}	3.6×10^{-8}	2.9×10^{-6}
Heptachlor epoxide	1.2×10^{-6}	7.6×10^{-7}	1.1×10^{-7}	2.0×10^{-6}
Total PCBs ^d	9.3×10^{-5}	3.1×10^{-5}	8.5×10^{-7}	1.3×10^{-4}
TOTAL LIFETIME INCREMENTAL CANCER RISK				1×10^{-4}

^a Noncancer risks based on developmental effects.

^b Noncancer risks based on immunologic effects.

^c Includes only the hazard index (HI) values for the immunologic effects of PCB 1248 PCB 1254. The other HIs are not additive with these values. A total HI value of 0.6 also results, with rounding, if additivity is assumed for the effects of methoxychlor and the developmental effects of PCB 1248. The health effects of 2-nitroaniline are not additive with the effects of any other Production Area COPC.

^d In accordance with USEPA Region I policy, risk of total PCBs was estimated assuming that all PCBs have the same cancer potency as PCB1260. This policy is not consistent with toxicological data which indicate that PCB 1248 and PCB 1254 are noncarcinogenic. PCB 1248 and PCB 1254 are the only PCBs detected in Warwick Area soil.

Section A8

Uncertainties

A8.0 Uncertainties

One of the primary objectives of the Risk Assessment is to characterize and quantify potential risks. The very nature of risk, being comprised of probability statements, connotes that uncertainty is involved. The fact that potential risks in the Risk Assessment are called "potential" accentuates the associated uncertainty because the risks evaluated do not exist at this time. In addition, there are uncertainties associated with the COPC selection process, future land-use scenarios, transport models, exposure input values, toxicity values, and the risk characterization process.

A8.1 COPC Selection Process

The COPC were selected using a screening process described by the USEPA in the HHM. While the method is useful for screening, it is based on oral toxicity values and does not address chemical-specific differences to such variables as environmental contaminant transport, dermal absorption rates, and toxicities via exposure routes other than ingestion.

A8.2 Future Land-Use Scenarios

Future land use for the Production Area is assumed to be a City of Cranston parking lot and storage facility for road salt, sand, and snow removal equipment. This is based on the plans of the City of Cranston (1995) and Ciba. This assumed future land use has a relatively high level of certainty. However, because the assumption was made in calculating potential risks that the Production Area would not be paved or in any way covered, the Risk Assessment greatly exaggerates exposure to contaminated soil, and thus, greatly overestimates potential cancer risks and noncancer hazards.

Unrestricted residential land use is conservatively assumed for the Warwick Area because there is uncertainty as to the future use of the land. Future industrial or commercial use of the Warwick Area is regarded as very plausible. The assumption of residential land use would likely overestimate the exposure associated with an industrial or commercial land-use scenario.

A8.3 Transport Models

Soil-to-air transport models were used in the Risk Assessment to predict concentrations of COPC in the air on-site that may be attributable to each of the site areas (Attachment 4). These models were intentionally selected and used in a manner that would tend to overestimate potential exposures of people. For example, a simple event of neutral stability, a mean annual

wind speed, and a constant worst-case wind direction were assumed conditions. Also, it was assumed that the soil surface contained no hardened crust. These are unrealistic assumptions which, together, exaggerate wind dispersion of soils. Although this approach results in overestimated potential exposures, it also allows for determining if more time-consuming efforts are necessary for the PHERE. Obviously, if these models show no significant contribution to unacceptable risks, as is the case in this study, then resources can be focused elsewhere in preparing a more site-specific, comprehensive PHERE.

A8.4 Exposure Assumption Values

Exposure assumption values used in the exposure assessment are generally regarded as overestimates of the "true" values. The HHEM advocates a "reasonable maximum exposure" (RME) approach to exposure assessment. The RME does not assume "worst-case" values for each exposure assumption value. However, the RME values recommended by the HHEM, such as contact rates, exposure frequencies, and exposure duration, are decidedly conservative (e.g., 95th percentile UCLs of possible values). The Risk Assessment basically followed the HHEM approach, using assumption values that were reviewed by Region I in the May, 1994, meeting and discussed during subsequent meetings and teleconferences. A few are somewhat less conservative than the default RME values which appear in the HHEM. Although there is uncertainty associated with every selected value, a few of these exposure variables are highlighted in the following paragraphs.

Maximum detected and 95th percentile UCL of the mean concentrations were used as the chemical concentration values. These are overestimates of average values. It is noted that concentrations that were qualified as estimated values during data validation ("J values") were also used in the Risk Assessment to derive the concentration values; nondetected values were assumed to be one-half the sample quantitation limits (SQL). These practices are consistent with the HHEM. The use of "J values" may result in either an overestimate or underestimate of actual average concentrations. Because many of the "J values" are less than one-half their respective SQLs, the assumption that a concentration equal to one-half the SQL is present, tends to overestimate actual average concentrations.

The soil ingestion rates (IR_s) are considered overestimates of actual values. The soil ingestion rate IR_s used in the exposure assessment for the on-site residential scenario is 200 mg/day for young children and 100 mg/day for older children and adults, as suggested in the HHEM. An ingestion rate of 50 mg/(work)day of soil was assumed for the on-site worker scenario.

However, in studies by Calabrese, et al., (1989) using 64 subjects, the median of the range for daily soil ingestion by young children (ages 1 through 4 years old) was found to be 9 to 40 mg/kg per day, depending on the tracer element used for the study. Work cited in the Exposure Factors Handbook (USEPA, 1990a) suggests that individuals 5 years of age and older ingest on average approximately 10 mg of soil per day. Soil ingestion for the residential scenario and contact were assumed to be proportionate to the amount of time spent at the Site. This is an overestimate, especially for adults, since one of the primary sources of ingested soil is associated with food.

The exposed body surface area values (SA_s) used in the Risk Assessment for the adult worker (5,000 cm^2), adult resident (5,000 cm^2), and child resident (2,000 cm^2) are overestimates. These values approximate 25 percent of the total body surface area and represent a person wearing a short-sleeved shirt, shorts, and shoes. Exposed areas using these SA_s values include the head, neck, hands, forearms, and lower legs. The adult worker used in the Risk Assessment is assumed to be at the Site only during the winter. Obviously, given the harsh Rhode Island winters, this worker would not dress in such attire, but would likely wear gloves, a hat, and several layers of clothing covering the body, including the arms and legs. The only areas left uncovered would be part of the face and possibly the neck. Thus, the true body surface area of the on-site worker potentially exposed to soil would be substantially less than 1,000 cm^2 . Regarding the on-site resident, a short-sleeved shirt, shorts and shoes may be reasonable attire for the summer months, but not during the spring and fall which comprise most of the annual exposure period. More reasonable SA_s values for residential exposure might be 3,000 cm^2 for an adult and 1,400 cm^2 for a young child.

The exposure duration used for the on-site residential scenario and on-site worker are 30 and 25 years, respectively. Few individuals work at the same location with the same job for as long as 25 years. The on-site residential scenario exposure duration is far greater than the median duration time of 9 years that an individual typically lives at a residence as referenced in the HHEM. These conservative exposure values, when combined, may overestimate the potential risk by two orders of magnitude over more realistic exposure assumptions, depending on the exposure scenario and the exposure values selected. This does not include the overestimations of toxicity discussed in Section A8.5.

A8.5 Toxicity Assessment

Uncertainties pertaining to the toxicity assessment are discussed in Section A6.0. These include uncertainties regarding development of the health effects criteria values, the classification of carcinogenicity, the extrapolation of exposure route-specific toxicity values to other routes of exposure, and the extrapolation of toxic effects observed in animal studies to potential adverse health effects in people. A summary of these uncertainties is provided in the following paragraphs.

The development of health effects criteria for noncancer health effects involves professional judgement. Depending on the nature of the toxicity studies, a safety factor of up to nearly four orders of magnitude may be built into the RfD or RfC value.

The USEPA weight-of-evidence classification system for carcinogens is used to examine and classify chemical agents with respect to their human toxicity. Most compounds that the USEPA classifies as carcinogens, including the COPC examined in the Risk Assessment, are B2 carcinogens. The carcinogenicity of these chemicals is based on animal data. There is uncertainty as to the nature of the carcinogenic response in humans, if any. Also, the mathematical models used to extrapolate from relatively high-dose rodent studies to relatively low-dose human exposures are the subject of much controversy. The approach taken by USEPA of almost exclusively using the linearized multistage model, combined with other assumptions, tends to overestimate potential ILCR. The USEPA is currently revising its carcinogen policies. The revised policies are to be enacted during 1995 or 1996. These could potentially impact the Risk Assessment.

When a noncancer or cancer health effects criterion was not available for a given route of exposure, the criterion from another route of exposure was adopted for use. This practice adds uncertainty and may either overestimate or underestimate toxicity.

A provisional RfD was derived for PCB 1248 because the USEPA has not established an RfD. An uncertainty factor of 10 was used in the estimation of a NOAEL for a LOAEL observed in a study on rhesus monkeys. An uncertainty factor of 3 may be justifiable, except the investigators of this critical study omitted key information about reproductive performance in the control group and their rhesus colony in general (see Section A6.2.1.1).

Total PCBs was used in the risk characterization, using the Region I policy assumption that the

combination of all PCBs is equal in cancer potency to PCB 1260 (USEPA 1995b). This practice is not consistent with PCBs toxicity data. A large toxicity database exists for PCB 1254, from which it is concluded that it is not carcinogenic. Also, existing studies suggest that PCB 1248 is not carcinogenic. Since most of the PCBs at the Site are PCB 1254 and PCB 1248, to assume that these mixtures are carcinogens with the same cancer potency as PCB 1260 grossly overestimates potential cancer risks.

A8.6 Risk Characterization

Uncertainty inherent to the risk characterization process involves the additivity assumption of adverse health effects associated with different chemicals. Chemicals in combination may act additively, antagonistically, synergistically, or not influence each other at all. Antagonistic relationships result in health effects that are less than those predicted by a chemical given alone; synergistic relationships result in health effects that exceed the results predicted by a chemical given alone and additivity of chemicals with similar effects. Therefore, the assumptions of additivity used in the Risk Assessment may either overestimate or underestimate human health risks.

The conservativeness of health effects criteria are discussed in Section A7.5. This conservativeness is compounded in the risk characterization process where multiple conservative values are combined together. This tends to exaggerate potential risks. Also, as discussed in Section A8.5, the Region I assumption that total PCBs have the same cancer potency as PCB 1260 can grossly overestimate potential cancer risks.

Section A9

Proposed Media Protection Standards

A9.0 Proposed Media Protection Standards

The Risk Assessment provides estimates of potential risks for the Cranston Site using the conservative guidance provided during the May, 1994, meeting with Region I and subsequent meetings and teleconferences. That is, the approach taken is biased towards overestimating risk. For example, all of the risk estimates are based on calculations using the 95 percent UCL of mean chemical concentrations instead of the actual mean. Even with this conservative approach, neither the Production Area nor the Warwick Area is predicted to pose an unacceptable potential risk. This was found in spite of the biased sampling approach used in the field investigations that targeted highly localized areas of suspected contamination.

PCBs are widespread in the Production Area as evidenced by the 89 percent frequency of detection for PCB 1254 and 39 percent for the PCB 1248 in surface soil samples. The risk assessment model for the on-site worker scenario (that is a combination of all the exposure assumption values and environmental transport models used in the risk assessment) can be used to estimate risk-based MPS values for PCB 1248 and PCB 1254. This is accomplished by beginning with the target THI value of 1 and "back-calculating" through the risk assessment model to the respective surface soil concentrations. Even though target risks are not exceeded, such estimated MPS values can be used to compare with the highest concentrations in the Production Area to determine if there are specific zones where PCB concentrations are higher than the MPSs. The estimated MPS value is 50 mg/kg for both PCB 1248 and 1254 using the approach described above. Potential additivity of PCB health effects are taken into account in these estimated MPSs.

A similar approach to estimating MPS values can be taken for the Warwick Area even though PCBs are obviously not as widespread there as in the Production Area with a frequency of detection for PCB 1248 in surface soil of 9 percent and for PCB 1254 of 47 percent. Also, most of the attention in the field investigation was concentrated on Solid Waste Management Unit No. 5 (SWMU-5). It contains a highly localized remnant of dredge materials from Pawtuxet River sediments taken from a waste water outfall and Coffey Dam area immediately adjacent to the Ciba Facility. Therefore, surface soil MPS values specific to the residential scenario for the Warwick Area would be useful to compare to PCB concentrations found in SWMU-5. The risk assessment model for the residential scenario is used to "back-calculate" MPSs for the PCBs. The estimated surface soil MPS is 9 mg/kg for both PCB 1248 and PCB 1254. Potential additive toxicity is accounted for in these estimates.

This Risk Assessment shows that corrective actions are not necessary for the Production and Warwick Areas solely on the basis of unacceptable potential risk to public health. However, Ciba may find it desirable to conduct some limited remediation in these Site areas for reasons other than potential risk, such as, facilitating productive use of the areas. Therefore, the risk-based total PCB surface soil MPS proposed for the Production Area is 50 mg/kg, and for the Warwick Area is 9 mg/kg.

With regard to the Production Area, Ciba has identified a zone where soil concentrations of PCBs are consistently above 50 mg/kg. However, the presence of this zone of PCB contamination is not a realistic public exposure concern because of the proposed use of this property as a paved vehicle parking facility. Thus, there would be virtually no exposure to this soil. The location of this zone is illustrated in Figure 3-1 in the main body of the Interim Remedial Measures Work Plan of which this Risk Assessment is an appendix. A clean-up level of 45 mg/kg (5 mg/kg lower than that allowed by the risk-based MPS) will be targeted for the Production Area to ensure that the average residual PCB concentration is below the 50 mg/kg limit.

Similarly, SWMU-5 (Figure A1-1) in the Warwick Area has soil PCB concentrations that are consistently above the 9 mg/kg MPS. This area is not a realistic exposure concern for the residential scenario because of the highly localized nature of this PCB contamination. The decision was made to reduce the target clean-up level in the Warwick Area to 1 mg/kg to allow for unrestricted use based on draft USEPA guidance (Disposal of Polychlorinated Biphenyls; Proposed Rule, December 12, 1994).



Section A10

References

A10.0 References

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Attachment 1

Analytical Summary and Statistical Analyses of Detected Chemicals

Production Area - Surface Soil

TABLE A1-1
ANALYTICAL SUMMARY OF DETECTED COMPOUNDS
PRODUCTION AREA
SURFACE SOIL

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) ^c
1,2-DICHLOROBENZENE	2	41	4.88	1.254	1.832	1.736	0.120	0.120	0.120
1,4-DICHLOROBENZENE	1	41	2.44	1.268	1.825	1.748	0.240	0.240	0.240
2,4,5-TP (SILVEX)	1	28	3.57	0.009	0.001	9.29E-03	6.00E-03	6.00E-03	6.00E-03
2,4-DIMETHYLPHENOL	1	41	2.44	1.274	1.822	1.753	0.110	0.110	0.110
2-METHYLNAPHTHALENE	4	41	9.76	1.263	1.828	1.744	0.380	0.038	0.380
2-NITROANILINE	8	41	19.51	6.249	9.061	8.632	0.890	0.044	0.890
3&4-METHYLPHENOL	5	16	31.25	0.179	0.166	0.252	0.770	0.023	0.252
4,4'-DDD	1	43	2.33	0.040	0.107	0.067	0.003	0.003	0.003
4-CHLOROANILINE	5	41	12.20	1.324	1.794	1.796	0.640	0.045	0.640
4-METHYLPHENOL	1	25	4.00	1.970	2.058	2.674	0.240	0.240	0.240
ACENAPHTHENE	10	41	24.39	1.235	1.843	1.720	0.210	0.057	0.210
ACENAPHTHYLENE	5	41	12.20	1.257	1.831	1.739	0.180	0.043	0.180
ACETOPHENONE	1	41	2.44	1.272	1.823	1.752	0.048	0.048	0.048
ALDRIN	1	43	2.33	0.030	0.061	4.59E-02	3.50E-03	3.50E-03	3.50E-03
ALPHA-BHC	2	43	4.65	0.030	0.061	4.60E-02	9.90E-03	1.60E-03	9.90E-03
ALPHA-CHLORDANE	2	43	4.65	0.030	0.061	4.61E-02	9.70E-03	2.50E-03	9.70E-03
AMMONIA AS N	9	25	36.00	0.761	1.059	1.123	5.300	0.290	1.123
ANILINE	1	41	2.44	1.277	1.820	1.755	0.230	0.230	0.230
ANTHRACENE	24	41	58.54	0.881	1.564	1.292	1.600	0.034	1.292
ARSENIC	29	32	90.63	9.011	21.409	15.434	125.000	0.520	15.434
BARIUM	31	31	100.00	46.535	31.698	56.197	106.000	4.600	56.197
BENZO(A)ANTHRACENE	28	41	68.29	1.144	1.413	1.516	3.100	0.150	1.516
BENZO(A)PYRENE	27	41	65.85	1.292	1.524	1.693	3.100	0.024	1.693
BENZO(B)FLUORANTHENE	30	41	73.17	1.568	1.562	1.979	4.300	0.027	1.979
BENZO(G,H,I)PERYLENE	21	41	51.22	1.351	1.745	1.810	2.900	0.130	1.810
BENZO(K)FLUORANTHENE	27	41	65.85	1.504	1.693	1.949	5.500	0.074	1.949
BERYLLIUM	30	31	96.77	0.400	0.176	0.454	0.730	0.090	0.454
BICARBONATE ALKALINITY	20	25	80.00	1382.000	1679.016	1956.559	7200.000	150.000	1956.559
BIS(2-CHLOROETHYL)ETHER	1	41	2.44	1.279	1.820	1.757	0.680	0.680	0.680
BIS(2-ETHYLHEXYL)PHTHALATE	16	41	39.02	1.094	1.490	1.486	4.100	0.061	1.486
BUTAZOLIDIN	1	25	4.00	9.978	10.017	13.406	5.200	5.200	5.200
BUTYLBENZYLPHTHALATE	13	41	31.71	1.834	5.285	3.224	33.000	0.042	3.224
CADMIUM	18	31	58.06	0.648	0.742	0.874	3.900	0.280	0.874
CALCIUM	26	26	100.00	20713.962	19653.293	27297.153	58500.00	207.000	27297.153

Production Area - Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) *	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) ^c
CARBONATE ALKALINITY	14	25	56.00	1248.680	1800.492	1864.809	7300.000	392.000	1864.809
CATION EXCHANGE CAPACITY	25	25	100.00	6.252	3.034	7.290	13.000	1.200	7.290
CHLORIDE	11	25	44.00	35.320	39.613	48.876	140.000	10.000	48.876
CHLOROBENZENE	7	40	17.50	0.203	1.022	0.477	0.280	0.032	0.280
CHLOROFORM	1	40	2.50	0.196	1.023	0.470	0.034	0.034	0.034
CHROMIUM	29	31	93.55	11.130	8.312	13.664	30.700	0.900	13.664
CHRYSENE	28	41	68.29	1.243	1.417	1.616	3.300	0.150	1.616
COBALT	29	31	93.55	3.039	1.307	3.437	6.000	0.380	3.437
COPPER	29	31	93.55	17.618	16.708	22.710	76.100	3.700	22.710
CYANIDE	11	34	32.35	1.323	2.261	1.981	12.600	0.560	1.981
DELTA-BHC	1	43	2.33	0.030	0.061	4.59E-02	2.40E-03	2.40E-03	2.40E-03
DI-N-BUTYLPHTHALATE	8	41	19.51	0.999	1.640	1.430	1.300	0.045	1.300
DIBENZ(A,H)ANTHRACENE	10	41	24.39	1.232	1.845	1.717	0.680	0.046	0.680
DIBENZOFURAN	12	41	29.27	1.200	1.862	1.689	0.130	0.035	0.130
DIMETHYLPHTHALATE	1	41	2.44	1.213	1.811	1.689	0.250	0.250	0.250
DINOSEB	3	40	7.50	0.065	0.079	0.086	0.009	0.002	0.009
ENDRIN ALDEHYDE	1	43	2.33	0.059	0.115	0.088	0.001	0.001	0.001
ETHYLBENZENE	10	40	25.00	1.287	7.900	3.407	50.000	0.006	3.407
FAMPHUR	2	42	4.76	0.147	0.040	0.158	0.016	0.006	0.016
FLUORANTHENE	33	41	80.49	1.599	1.734	2.055	8.400	0.051	2.055
FLUORENE	12	41	29.27	1.207	1.858	1.695	0.180	0.048	0.180
GAMMA-BHC	2	43	4.65	0.030	0.061	0.046	0.003	0.003	0.003
GAMMA-CHLORDANE (d)	7	43	16.28	0.066	0.257	0.132	1.700	0.008	0.132
HEPTACHLOR	1	43	2.33	0.030	0.061	4.56E-02	1.40E-02	1.40E-02	1.40E-02
INDENO(1,2,3-CD)PYRENE	21	41	51.22	1.325	1.761	1.789	2.300	0.045	1.789
IRGASAN DP-300	3	26	11.54	9.417	10.135	12.812	4.200	0.570	4.200
IRON	26	26	100.00	10472.308	4028.158	11821.605	21300.00	3390.000	11821.605
KEPONE	1	43	2.33	0.127	0.567	0.272	0.015	0.015	0.015
LEAD	29	31	93.55	54.308	82.536	79.464	378.000	3.600	79.464
M&P-XYLENE	27	40	67.50	10.082	63.233	27.048	400.000	0.006	27.048
MAGNESIUM	26	26	100.00	2114.923	1270.301	2540.431	5360.000	158.000	2540.431
MANGANESE	26	26	100.00	166.935	66.563	189.231	359.000	42.900	189.231
MERCURY	22	30	73.33	0.460	0.485	0.610	1.600	0.110	0.610
METHOXYCHLOR	7	43	16.28	0.354	0.783	0.555	3.600	0.120	0.555
METHYL PARATHION	2	42	4.76	0.009	0.001	8.87E-03	6.50E-03	5.60E-03	6.50E-03
METHYLENE CHLORIDE	6	40	15.00	0.269	1.033	0.546	0.010	0.005	0.010
NAPHTHALENE	14	41	34.15	0.830	1.654	1.265	0.680	0.033	0.680
NICKEL	28	31	90.32	7.774	5.722	9.518	26.600	1.500	9.518
NITRATE-NITRITE AS N	22	25	88.00	1.938	2.020	2.629	7.900	0.160	2.629
NITROBENZENE	3	41	7.32	1.253	1.833	1.735	0.140	0.081	0.140
O-XYLENE	19	40	47.50	3.034	18.968	8.124	120.000	0.009	8.124

Production Area - Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) ^c
OCDD	1	5	20.00	0.001	0.000	6.24E-04	4.60E-04	4.60E-04	4.60E-04
ORTHOPHOSPHATE	20	25	80.00	5.155	8.709	8.135	43.000	0.710	8.135
PCB-1248 (d)	38	98	38.78	0.29 (e)	NA (f)	0.44 (g)	4500.000	0.020	0.440
PCB-1254 (d)	95	107	88.79	2.40 (e)	NA (f)	3.60 (g)	84.000	0.043	3.600
PCB-1260 (d)	7	52	13.46	4.650	27.657	11.109	6.100	0.130	6.1
PERCENT MOISTURE	20	20	100.00	8.300	4.777	10.147	21.000	0.000	10.147
PH	12	12	100.00	8.458	1.481	9.226	12.000	6.400	9.226
PHENANTHRENE	28	41	68.29	1.147	1.490	1.539	5.000	0.093	1.539
PHENOL	1	41	2.44	1.277	1.820	1.756	0.630	0.630	0.630
POTASSIUM	25	26	96.15	841.865	292.108	939.712	1260.000	389.000	939.712
PYRENE	32	41	78.05	1.819	1.838	2.303	6.700	0.061	2.303
SODIUM	13	26	50.00	150.623	78.024	176.759	329.000	90.200	176.759
STYRENE	2	40	5.00	0.196	1.023	0.470	0.049	0.039	0.049
SULFATE	14	25	56.00	111.900	119.742	152.876	400.000	29.000	152.876
SULFIDE	5	25	20.00	22.540	12.973	26.979	66.000	14.000	26.979
SULFOTEP	1	42	2.38	0.021	0.011	2.33E-02	9.40E-03	9.40E-03	9.40E-03
TCDF	3	5	60.00	0.000	0.000	1.77E-04	1.90E-04	9.50E-05	1.77E-04
TETRACHLOROETHENE	1	40	2.50	0.197	1.022	0.471	0.069	0.069	0.069
THALLIUM	1	29	3.45	0.198	0.053	0.214	0.260	0.260	0.214
TIN	1	26	3.85	5.185	4.214	6.596	25.600	25.600	6.596
TINUVIN 327	1	24	4.17	9.269	9.570	12.617	5.200	5.200	5.200
TINUVIN 328	3	3	100.00	4.533	1.721	7.435	5.900	2.600	5.900
TOLUENE	18	40	45.000	0.393	1.198	0.714	4.600	0.007	0.714
TOTAL ALKALINITY	24	25	96.00	2744.720	2633.962	3646.062	10000.00	150.000	3646.062
TOTAL ORGANIC CARBON	25	25	100.00	2137.600	2596.859	3026.245	9200.000	0.000	3026.245
TRCDF	2	5	40.00	0.226	0.331	0.541	0.730	0.400	0.541
TRICHLOROFLUOROMETHANE	3	40	7.50	0.413	2.124	0.983	0.330	0.071	0.330
VANADIUM	28	31	90.32	14.981	18.947	20.756	108.000	1.400	20.756
ZINC	31	31	100.00	183.606	184.509	239.843	759.000	13.000	239.843

- Arithmetic mean concentration, unless otherwise indicated.
- 95th percentile upper confidence limit of the arithmetic mean concentration, unless otherwise indicated.
- Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
- Shading indicates that the chemical was selected as a chemical of potential concern.
- Geometric mean concentration.
- NA - Not applicable to a lognormal distribution.
- 95th percentile upper confidence limit of the geometric mean concentration.

Production Area - Combined Soil

TABLE A1-2
ANALYTICAL SUMMARY OF DETECTED COMPOUNDS
PRODUCTION AREA
COMBINED SOIL

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)
1,2,4-TRICHLOROBENZENE	3	84	3.57	0.862	1.454	1.127	0.640	0.190	0.640
1,2-DICHLOROBENZENE	3	84	3.57	0.857	1.456	1.122	0.760	0.120	0.760
1,4-DICHLOROBENZENE	1	84	1.19	0.860	1.454	1.125	0.240	0.240	0.240
1,4-DIOXANE	1	84	1.19	2.790	2.615	3.267	4.000	4.000	3.267
2,4,5-T	2	48	4.17	0.011	0.001	0.011	0.017	0.006	0.011
2,4,5-TP (SILVEX)	2	48	4.17	0.009	0.001	9.25E-03	7.20E-03	6.00E-03	7.20E-03
2,4-D	6	48	12.50	0.059	0.018	0.064	0.110	0.008	0.064
2,4-DICHLOROPHENOL	4	84	4.76	0.971	1.548	1.253	6.200	2.600	1.253
2,4-DIMETHYLPHENOL	7	84	8.33	0.885	1.457	1.150	1.700	0.080	1.150
2,6-DICHLOROPHENOL	1	84	1.19	0.891	1.468	1.159	2.800	2.800	1.159
2-BUTANONE	4	80	5.00	1.414	6.601	2.647	0.300	0.130	0.300
2-METHYLNAPHTHALENE	5	84	5.95	0.857	1.456	1.122	0.380	0.038	0.380
2-METHYLPHENOL	1	84	1.19	0.863	1.454	1.128	0.084	0.084	0.084
2-NITROANILINE	9	84	10.71	4.301	7.173	5.609	4.200	0.044	4.200
2-NITROPHENOL	1	84	1.19	0.863	1.454	1.128	0.075	0.075	0.075
3&4-METHYLPHENOL	17	42	40.48	0.282	0.343	0.372	1.200	0.023	0.372
3,3'-DICHLOROBENZIDINE	1	84	1.19	1.725	2.861	2.246	0.730	0.730	0.730
4,4'-DDD	2	86	2.33	0.026	0.080	0.041	0.230	0.003	0.041
4,4'-DDE	1	86	1.16	0.031	0.107	0.051	0.710	0.710	0.051
4,4'-DDT	2	86	2.33	0.039	0.092	0.055	0.350	0.084	0.055
4-CHLOROANILINE	8	84	9.52	0.917	1.422	1.177	0.640	0.042	0.640
4-METHYLPHENOL	4	42	9.52	1.559	1.863	2.043	3.400	0.100	2.043
ACENAPHTHENE	12	84	14.29	0.840	1.462	1.107	0.280	0.049	0.280
ACENAPHTHYLENE	5	84	5.95	0.855	1.457	1.120	0.180	0.043	0.180
ACETONE	1	80	1.25	1.409	6.602	2.642	0.053	0.053	0.053
ACETOPHENONE	4	84	4.76	0.870	1.451	1.135	0.660	0.048	0.660
ALDRIN	2	86	2.33	0.023	0.064	0.034	0.440	0.004	0.034
ALPHA-BHC	5	86	5.81	0.018	0.045	0.027	0.018	0.001	0.018
ALPHA-CHLORDANE	2	86	2.33	0.018	0.045	0.026	0.010	0.003	0.010
AMMONIA AS N	20	42	47.62	1.548	2.230	2.127	8.500	0.170	2.127
ANILINE	5	84	5.95	0.867	1.452	1.131	0.280	0.210	0.280
ANTHRACENE	32	84	38.10	0.659	1.258	0.889	1.600	0.034	0.889
ARSENIC	52	55	94.55	7.770	16.440	11.503	125.000	0.520	11.503
BARIUM	52	52	100.00	36.973	27.889	43.486	106.000	4.600	43.486
BENZO(A)ANTHRACENE	41	84	48.81	0.797	1.192	1.015	3.100	0.140	1.015

Production Area - Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) *	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)
BENZO(A)PYRENE	44	84	52.38	0.872	1.277	1.104	3.100	0.024	1.104
BENZO(B)FLUORANTHENE	52	84	61.90	1.034	1.351	1.280	4.300	0.027	1.280
BENZO(G,H,I)PERYLENE	30	84	35.71	0.910	1.415	1.168	2.900	0.086	1.168
BENZO(K)FLUORANTHENE	42	84	50.00	0.970	1.418	1.228	5.500	0.036	1.228
BERYLLIUM	49	52	94.23	0.385	0.174	0.425	0.790	0.090	0.425
BETA-BHC	1	86	1.16	0.019	0.046	0.028	0.100	0.100	0.028
BICARBONATE ALKALINITY	33	42	78.57	925.988	1408.408	1291.958	7200.000	80.000	1291.958
BIS(2-CHLOROETHYL)ETHER	2	84	2.38	0.864	1.454	1.129	0.680	0.050	0.680
BIS(2-ETHYLHEXYL)PHTHALATE	36	84	42.86	0.850	1.228	1.074	4.100	0.054	1.074
BUTAZOLIDIN	1	40	2.50	7.858	9.108	10.301	5.200	5.200	5.200
BUTYLBENZYLPHTHALATE	18	84	21.43	1.130	3.781	1.819	33.000	0.042	1.819
CADMIUM	21	54	38.89	0.473	0.598	0.610	3.900	0.240	0.610
CALCIUM	43	43	100.00	13841.372	17808.579	18414.750	58500.000	104.000	18414.750
CARBONATE ALKALINITY	20	42	47.62	904.179	1495.679	1292.826	7300.000	60.000	1292.826
CATION EXCHANGE CAPACITY	42	42	100.00	5.345	2.956	6.113	13.000	1.200	6.113
CHLORIDE	21	42	50.00	52.393	65.242	69.346	260.000	10.000	69.346
CHLOROBENZENE	9	80	11.25	0.666	3.240	1.271	0.280	0.018	0.280
CHLOROBENZILATE	1	85	1.18	0.813	1.511	1.087	0.098	0.098	0.098
CHLOROFORM	1	80	1.25	0.664	3.240	1.269	0.034	0.034	0.034
CHROMIUM	52	54	96.30	10.230	6.956	11.825	30.700	0.600	11.825
CHRYSENE	43	84	51.19	0.859	1.200	1.078	3.300	0.052	1.078
COBALT	49	52	94.23	3.486	1.825	3.913	8.600	0.350	3.913
COPPER	52	54	96.30	15.518	14.381	18.813	76.100	0.810	18.813
CYANIDE	14	59	23.73	1.441	2.721	2.037	13.600	0.560	2.037
DELTA-BHC	2	86	2.33	0.019	0.046	0.028	0.086	0.002	0.028
DI-N-BUTYLPHTHALATE	14	84	16.67	0.715	1.316	0.955	1.300	0.042	0.955
DIBENZ(A,H)ANTHRACENE	12	84	14.29	0.809	1.470	1.077	0.680	0.046	0.680
DIBENZOFURAN	14	84	16.67	0.821	1.470	1.089	0.400	0.035	0.400
DIETHYLPHTHALATE	1	84	1.19	0.806	1.360	1.054	0.670	0.670	0.670
DIMETHYLPHTHALATE	1	84	1.19	0.833	1.439	1.096	0.250	0.250	0.250
DINOSEB	5	78	6.41	0.102	0.201	0.140	0.009	0.002	0.009
DISULFOTON	1	84	1.19	0.062	0.011	0.064	0.010	0.010	0.010
ENDOSULFAN SULFATE	1	86	1.16	0.072	0.134	0.096	0.096	0.096	0.096
ENDRIN ALDEHYDE	2	86	2.33	0.035	0.086	0.051	0.002	0.001	0.002
ETHYL PARATHION	1	84	1.19	0.029	0.013	0.032	0.013	0.013	0.013
ETHYLBENZENE	24	80	30.00	1.693	6.877	2.977	50.000	0.006	2.977
FAMPHUR	2	84	2.38	0.154	0.032	0.160	0.016	0.006	0.016
FLUORANTHENE	60	84	71.43	0.989	1.363	1.237	8.400	0.043	1.237
FLUORENE	17	84	20.24	0.820	1.471	1.088	0.180	0.047	0.180
GAMMA-BHC	9	86	10.47	0.019	0.045	0.027	0.036	0.002	0.027
GAMMA-CHLORDANE (d)	13	86	15.12	0.037	0.183	0.070	1.700	0.002	0.070

Production Area - Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) *	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)
HEPTACHLOR	4	86	4.65	0.018	0.045	0.026	0.042	0.004	0.026
INDENO(1,2,3-CD)PYRENE	31	84	36.90	0.874	1.426	1.134	2.300	0.045	1.134
IRGASAN DP-300	7	44	15.91	24.485	71.230	42.568	390.000	0.570	42.568
IRON	43	43	100.00	10929.884	5445.449	12328.316	29900.000	925.000	12328.316
ISODRIN	2	86	2.33	0.026	0.080	0.040	0.220	0.010	0.040
KEPONE	1	86	1.16	0.073	0.403	0.146	0.015	0.015	0.015
LEAD	50	52	96.15	37.207	66.765	52.798	378.000	3.600	52.798
M&P-XYLENE	48	80	60.00	9.443	47.247	18.270	400.000	0.006	18.270
MAGNESIUM	43	43	100.00	1973.558	1271.141	2299.997	5360.000	108.000	2299.997
MANGANESE	43	43	100.00	160.349	72.774	179.038	359.000	15.200	179.038
MERCURY	32	52	61.54	0.341	0.448	0.446	1.600	0.060	0.446
METHOXYCHLOR	12	86	13.95	0.208	0.574	0.311	3.600	0.032	0.311
METHYL PARATHION	6	84	7.14	0.009	0.001	8.76E-03	7.00E-03	5.60E-03	7.00E-03
METHYLENE CHLORIDE	20	80	25.00	0.791	3.271	1.402	1.500	0.005	1.402
N-OCTANE	2	5	40.00	3.263	5.226	8.246	12.000	4.300	8.246
NAPHTHALENE	29	84	34.52	0.549	1.199	0.767	0.680	0.029	0.680
NICKEL	49	54	90.74	8.156	5.607	9.441	26.600	0.630	9.441
NITRATE-NITRITE AS N	35	42	83.33	2.392	2.483	3.037	9.600	0.160	3.037
NITROBENZENE	5	84	5.95	0.873	1.462	1.140	2.100	0.081	1.140
O-XYLENE	37	80	46.25	2.609	13.854	5.198	120.000	0.009	5.198
OCDD	1	9	11.11	0.000	0.000	5.14E-04	4.60E-04	4.60E-04	4.60E-04
ORTHOPHOSPHATE	35	42	83.33	10.285	16.475	14.566	77.000	0.710	14.566
P-PHENYLENEDIAMINE	1	84	1.19	4.734	7.578	6.116	15.000	15.000	6.116
PCB-1248 (d)	38	141	26.95	0.150 (e)	NA (f)	0.210 (g)	4500.000	0.020	4500.000
PCB-1254 (d)	128	150	85.33	1.400 (e)	NA (f)	2.000 (g)	84.000	0.043	84.000
PCB-1260 (d)	15	95	15.79	2.857	20.515	6.374	13.000	0.070	6.374
PERCENT MOISTURE	39	39	100.00	8.374	4.620	9.630	21.000	0.000	9.630
PH	22	22	100.00	8.114	1.746	8.754	12.000	4.700	8.754
PHENANTHRENE	49	84	58.33	0.724	1.126	0.929	5.000	0.030	0.929
PHENOL	4	84	4.76	0.857	1.456	1.123	0.630	0.150	0.630
POTASSIUM	42	43	97.67	721.244	317.799	802.857	1260.000	73.000	802.857
PYRENE	58	84	69.05	1.193	1.550	1.476	6.700	0.042	1.476
SODIUM	23	43	53.49	152.714	84.149	174.324	350.000	29.500	174.324
STYRENE	5	80	6.25	0.664	3.240	1.269	0.049	0.008	0.049
SULFATE	26	42	61.90	161.798	312.621	243.031	1800.000	17.000	243.031
SULFIDE	6	42	14.29	20.500	10.714	23.284	66.000	14.000	23.284
SULFOTEPP	1	84	1.19	0.018	0.010	0.020	0.009	0.009	0.009
TCDF	4	9	44.44	0.000	0.000	1.31E-04	1.90E-04	5.60E-05	1.31E-04
TETRACHLOROETHENE	1	80	1.25	0.664	3.240	1.269	0.069	0.069	0.069
THALLIUM	1	48	2.08	0.196	0.054	0.209	0.260	0.260	0.209
TIN	1	43	2.33	4.851	3.297	5.698	25.600	25.600	5.698

Production Area - Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)
TINUVIN 327	2	40	5.00	7.187	8.635	9.504	5.200	0.490	5.200
TINUVIN 328	5	5	100.00	5.600	3.890	9.309	12.000	2.400	9.309
TOLUENE	31	80	38.75	30.822	164.400	61.536	1200.000	0.006	61.536
TOTAL ALKALINITY	38	42	90.48	1970.369	2368.800	2585.894	10000.000	87.000	2585.894
TOTAL ORGANIC CARBON	42	42	100.00	3388.000	2132.766	3942.192	9200.000	540.000	3942.192
TRCDF	3	10	30.00	0.140	0.252	0.286	0.730	0.270	0.286
TRICHLOROFLUOROMETHANE	3	80	3.75	1.336	6.606	2.570	0.330	0.071	0.330
VANADIUM	45	52	86.54	12.357	15.089	15.881	108.000	0.880	15.881
ZINC	51	54	94.44	130.719	158.038	166.936	759.000	2.200	166.936

- a. Arithmetic mean concentration, unless otherwise indicated.
- b. 95th percentile upper confidence limit of the arithmetic mean concentration, unless otherwise indicated.
- c. Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
- d. Shading indicates that the chemical was selected as a chemical of potential concern (COPC).
- e. Geometric mean concentration.
- f. NA - Not applicable to a lognormal distribution.
- g. 95th percentile upper confidence limit of the geometric mean concentration.

Warwick Area - Surface Soil

TABLE A1-3
ANALYTICAL SUMMARY OF DETECTED COMPOUNDS
WARWICK AREA
SURFACE SOIL

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) ^c
1,1-BIPHENYL	1	2	50.00	1.0375	1.2198	6.4833	1.9	1.9	1.9000
1,1-DICHLOROETHANE	1	32	3.13	0.0478	0.0303	0.0568	0.044	0.044	0.0440
1,2-DICHLOROBENZENE	1	31	3.23	1.3803	2.1297	2.0294	0.18	0.18	0.1800
1,4-DICHLOROBENZENE	2	31	6.45	1.3865	2.1281	2.0351	0.039	0.032	0.0390
2,4,5-T	1	31	3.23	0.0121	0.0070	0.0143	0.0095	0.0095	0.0095
2,4,5-TP (SILVEX)	4	31	12.90	0.0314	0.0698	0.0527	0.34	0.02	0.0527
2-BUTANONE	2	32	6.25	0.1031	0.0637	0.1222	0.23	0.18	0.1222
2-METHYLNAPHTHALENE	6	31	19.35	1.3441	2.1482	1.9989	0.36	0.014	0.3600
2-NITROANILINE (d)	2	31	6.45	7.1542	10.6697	10.4062	7	0.98	7.0000
3&4-METHYLPHENOL	1	6	16.67	0.1520	0.0640	0.2046	0.022	0.022	0.0220
3,3'-DIMETHYLBENZIDINE	1	31	3.23	2.9161	4.3040	4.2279	6.6	6.6	4.2279
4,4'-DDD	2	32	6.25	0.0654	0.2290	0.1341	0.41	0.018	0.1341
4,4'-DDE	6	33	18.18	0.0774	0.2414	0.1487	0.65	0.004	0.1487
4,4'-DDT	9	32	28.13	0.1185	0.4461	0.2523	0.51	0.00096	0.2523
4-CHLOROANILINE	10	31	32.26	1.4424	1.8587	2.0089	7.4	0.31	2.0089
ACENAPHTHENE	3	31	9.68	1.3716	2.1350	2.0223	0.16	0.016	0.1600
ACENAPHTHYLENE	3	31	9.68	1.3864	2.1281	2.0350	0.11	0.061	0.1100
ACETONE	1	32	3.13	0.1047	0.0700	0.1257	0.32	0.32	0.1257
ALDRIN (d)	3	33	9.09	0.1120	0.3252	0.2080	1.1	0.13	0.2080
ALPHA-BHC	6	33	18.18	0.0949	0.2965	0.1825	1.2	0.001	0.1825
ALPHA-CHLORDANE	3	32	9.38	0.0550	0.2201	0.1210	0.077	0.004	0.0770
AMMONIA AS N	5	25	20.00	0.9690	1.3063	1.4160	5.2	1.1	1.4160
ANTHRACENE	10	31	32.26	1.3073	2.1638	1.9668	0.32	0.031	0.3200
ANTIMONY	6	23	26.09	2.2654	8.6269	5.3540	41.8	0.86	5.3540
ARSENIC	27	27	100.00	9.0037	3.7988	10.2509	16.2	2.4	10.2509
BARIUM	31	31	100.00	113.6484	227.7039	183.0503	1270	7.3	183.0503
BENZENE	1	32	3.13	0.0474	0.0304	0.0565	0.034	0.034	0.0340
BENZO(A)ANTHRACENE	15	31	48.39	0.9723	1.2572	1.3554	1.6	0.14	1.3554
BENZO(A)PYRENE	13	31	41.94	1.1502	1.5519	1.6232	1.7	0.025	1.6232
BENZO(B)FLUORANTHENE	14	31	45.16	1.2336	1.3580	1.6475	2.8	0.042	1.6475
BENZO(G,H,I)PERYLENE	9	31	29.03	1.4471	2.1041	2.0884	1.2	0.064	1.2000
BENZO(K)FLUORANTHENE	13	31	41.94	1.3034	1.5083	1.7631	3.6	0.062	1.7631
BERYLLIUM (d)	31	31	100.00	0.622 (e)	NA (f)	0.718 (f)	2	0.32	0.7180
BETA-BHC	2	32	6.25	0.0523	0.2202	0.1183	0.0096	0.0091	0.0096
BICARBONATE ALKALINITY	17	25	68.00	230.9600	222.9096	307.2397	940	97	307.2397
BIS(2-CHLOROETHYL)ETHER	2	31	6.45	1.3797	2.1293	2.0287	0.43	0.33	0.4300

Warwick Area - Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) ^c
BIS(2-ETHYLHEXYL)PHTHALATE	14	31	45.16	10.4613	31.4433	20.0449	140	0.1	20.0449
BUTYLBENZYLPHthalATE	6	31	19.35	1.3891	2.1279	2.0377	0.78	0.046	0.7800
CADMIUM	15	31	48.39	1.5608	2.1672	2.2214	6.9	0.28	2.2214
CALCIUM	25	25	100.00	1465.8800	792.1142	1736.9415	3730	363	1736.9415
CATION EXCHANGE CAPACITY	25	25	100.00	7.1040	4.9712	8.8051	18	1.4	8.8051
CHLORIDE	13	25	52.00	71.7000	86.9734	101.4623	300	19	101.4623
CHLOROBEZENE	9	32	28.13	0.4073	0.8703	0.6684	3.6	0.0057	0.6684
CHLOROFORM	1	32	3.13	0.0479	0.0303	0.0570	0.048	0.048	0.0480
CHROMIUM	31	31	100.00	68.1548	107.8930	101.0396	357	1.3	101.0396
CHRYSENE	14	31	45.16	1.0750	1.2914	1.4686	2.3	0.12	1.4686
COBALT	27	27	100.00	4.4259	1.7819	5.0110	7.8	1	5.0110
COPPER	30	31	96.77	118.0161	351.7469	225.2251	1960	2.9	225.2251
CYANIDE	9	30	30.00	1.8620	2.7130	2.7035	9.4	0.8	2.7035
DELTA-BHC	3	33	9.09	0.0582	0.2199	0.1231	0.26	0.002	0.1231
DI-N-BUTYLPHthalATE	1	31	3.23	1.3780	2.1315	2.0276	0.057	0.057	0.0570
DI-N-OCTYLPHthalATE	8	31	25.81	2.5247	5.2476	4.1241	23	0.04	4.1241
DIBENZ(A,H)ANTHRACENE	3	31	9.68	1.3817	2.1308	2.0312	0.13	0.083	0.1300
DIBENZOFURAN	3	31	9.68	1.3598	2.1403	2.0122	0.2	0.052	0.2000
DIELDRIN (d)	5	32	15.63	0.0806	0.2667	0.1606	0.91	0.0019	0.1606
DIOSEB	1	31	3.23	0.0431	0.0675	0.0637	0.072	0.072	0.0637
DISULFOTON	3	30	10.00	0.0569	0.0246	0.0645	0.0077	0.0059	0.0077
ENDOSULFAN I	3	32	9.38	0.0524	0.2202	0.1185	0.018	0.01	0.0180
ENDOSULFAN II	1	32	3.13	0.1524	0.6613	0.3508	0.018	0.018	0.0180
ENDOSULFAN SULFATE	3	32	9.38	0.2464	1.0589	0.5640	0.29	0.0075	0.2900
ENDRIN	6	32	18.75	0.0702	0.2362	0.1411	0.54	0.0031	0.1411
ENDRIN ALDEHYDE	5	32	15.63	0.2206	0.7449	0.4441	3.5	0.0021	0.4441
ETHYL PARATHION	2	30	6.67	0.0359	0.0129	0.0399	0.0064	0.0056	0.0064
ETHYLBENZENE	1	32	3.13	0.0472	0.0305	0.0564	0.027	0.027	0.0270
FLUORANTHENE	17	31	54.84	1.2277	1.1463	1.5771	3.7	0.038	1.5771
FLUORENE	7	31	22.58	1.3418	2.1494	1.9969	0.23	0.035	0.2300
GAMMA-BHC	2	32	6.25	0.0518	0.2204	0.1179	0.0017	0.0014	0.0017
GAMMA-CHLORDANE	9	32	28.13	0.0783	0.2345	0.1487	0.5	0.004	0.1487
HEPTACHLOR	1	32	3.13	0.0518	0.2203	0.1179	0.0032	0.0032	0.0032
HEPTACHLOR EPOXIDE (d)	7	32	21.88	0.1016	0.3000	0.1916	1.2	0.0022	0.1916
INDENO(1,2,3-CD)PYRENE	8	31	25.81	1.4132	2.1159	2.0581	0.86	0.07	0.8600
IRON	25	25	100.00	12327.6000	3723.4228	13601.7553	20000	4610	13601.7553
ISODRIN	2	32	6.25	0.0769	0.2612	0.1553	0.85	0.0072	0.1553
KEPONE	1	32	3.13	0.0808	0.2343	0.1511	0.26	0.26	0.1511
LEAD	26	27	96.30	84.0056	102.6756	117.7160	428	2.8	117.7160
M&P-XYLENE	8	32	25.00	0.0456	0.0300	0.0546	0.07	0.0065	0.0546
MAGNESIUM	25	25	100.00	1713.5600	1030.6747	2066.2569	5360	290	2066.2569
MANGANESE	25	25	100.00	221.1360	68.3698	244.5321	416	83.4	244.5321

Warwick Area - Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) ^c
MERCURY	15	31	48.39	0.2261	0.2792	0.3112	0.94	0.13	0.3112
METHOXYCHLOR (d)	13	34	38.24	110.3893	417.4362	231.8771	2200	0.046	231.8771
METHYLENE CHLORIDE	3	32	9.38	0.2386	0.3910	0.3559	0.025	0.011	0.0250
N-NITROSO-DI-N-PROPYLAMINE	1	31	3.23	1.3684	2.1372	2.0198	0.17	0.17	0.1700
NAPHTHALENE	16	31	51.61	1.1290	1.4793	1.5799	3.5	0.036	1.5799
NICKEL	28	31	90.32	17.0097	34.6186	27.5611	199	2.7	27.5611
NITRATE-NITRITE AS N	22	25	88.00	1.5752	1.5060	2.0906	6.4	0.34	2.0906
NITROBENZENE	2	31	6.45	1.4774	2.1294	2.1264	2.9	0.48	2.1264
O-XYLENE	4	32	12.50	0.0445	0.0306	0.0537	0.046	0.017	0.0460
ORTHOPHOSPHATE	20	25	80.00	4.5704	6.7629	6.8847	29	0.36	6.8847
PCB-1248 (d)	3	34	8.82	6.8904	28.3977	15.1550	160	8.1	15.1550
PCB-1254 (d)	15	32	46.88	2.8607	7.7752	5.1932	36	0.032	5.1932
PERCENT MOISTURE	24	24	100.00	12.3333	8.2031	15.2033	28	0	15.2033
PH	15	15	100.00	6.1800	0.4931	6.4042	7.1	5.6	6.4042
PHENACETIN	1	31	3.23	1.4084	2.1187	2.0541	1	1	1.0000
PHENANTHRENE	17	31	54.84	0.8669	0.8585	1.1286	1.7	0.18	1.1286
PHENOL	3	31	9.68	1.1845	1.8565	1.7503	0.89	0.35	0.8900
PHORATE	1	30	3.33	0.0369	0.0112	0.0404	0.0096	0.0096	0.0096
POTASSIUM	25	25	100.00	767.4400	853.6258	1059.5508	4630	331	1059.5508
PROPACIN	1	25	4.00	8.2420	11.0038	12.0075	24	24	12.0075
PYRENE	18	31	58.06	1.2765	1.0767	1.6047	3	0.053	1.6047
SAFROLE	4	31	12.90	2.4648	5.2075	4.0520	28	0.7	4.0520
SODIUM	15	25	60.00	117.2300	60.5521	137.9509	217	118	137.9509
SULFATE	11	25	44.00	128.8200	199.8416	197.2058	890	13	197.2058
SULFIDE	5	25	20.00	92.1200	335.4909	206.9250	1700	35	206.9250
SULFOTEP	1	31	3.23	0.0241	0.0085	0.0267	0.0041	0.0041	0.0041
TETRACHLOROETHENE	9	32	28.13	0.1261	0.4190	0.2518	2.4	0.009	0.2518
THIONAZIN	1	30	3.33	0.2207	0.1157	0.2566	0.0058	0.0058	0.0058
TIN	6	31	19.35	7.4226	7.6452	9.7528	37.8	14.7	9.7528
TINUVIN 327	8	25	32.00	6.3408	8.5561	9.2687	18	0.57	9.2687
TOLUENE	18	32	56.25	0.2324	0.4102	0.3555	1.8	0.0069	0.3555
TOTAL ALKALINITY	17	25	68.00	230.9600	222.9096	307.2397	940	97	307.2397
TOTAL ORGANIC CARBON	25	25	100.00	11732.0000	11172.9005	15555.3666	28000	240	15555.3666
TRICHLOROETHENE	2	32	6.25	0.0497	0.0335	0.0597	0.13	0.041	0.0597
VANADIUM	19	27	70.37	10.5074	5.2704	12.2378	22	1.7	12.2378
ZINC	28	31	90.32	2538.6726	4505.7211	3911.9722	16100	24.8	3911.9722

- Arithmetic mean concentration.
- 95th percentile upper confidence limit of the arithmetic mean concentration.
- Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
- Shading indicates that the chemical was selected as a chemical of potential concern (COPC).

Project 1.003.06
February 16, 1995

Appendix A
Attachment 1, Table 3, page 3

WAR-SS.XLS

Warwick Area - Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) ^c
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e. Geometric mean concentration.

f. NA - Not applicable to a lognormal distribution.

g. 95th percentile upper confidence limit of the geometric mean concentration.

Warwick Area - Combined Soil

TABLE A1-4
ANALYTICAL SUMMARY OF DETECTED COMPOUNDS
WARWICK AREA
COMBINED SOIL

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) ^c
1,1-BIPHENYL	1	2	50.00	1.0375	1.2198	6.483	1.900	1.900	1.900
1,1-DICHLOROETHANE	1	52	1.92	0.0416	0.0617	0.056	0.044	0.044	0.044
1,2-DICHLOROBENZENE	1	41	2.44	1.0983	1.9124	1.601	0.180	0.180	0.180
1,4-DICHLOROBENZENE	2	41	4.88	1.1030	1.9118	1.606	0.039	0.032	0.039
2,4,5-T	1	38	2.63	0.0129	0.0095	0.015	0.010	0.010	0.010
2,4,5-TP (SILVEX)	4	38	10.53	0.0285	0.0636	0.046	0.340	0.020	0.046
2-BUTANONE	2	52	3.85	0.1147	0.2882	0.182	0.230	0.180	0.182
2-CHLOROPHENOL	1	41	2.44	1.1102	1.9079	1.612	0.240	0.240	0.240
2-METHYLNAPHTHALENE	11	41	26.83	1.0517	1.9336	1.560	0.360	0.010	0.360
2-NITROANILINE (d)	3	41	7.32	5.6817	9.6092	8.209	7.000	0.920	7.000
3&4-METHYLPHENOL	2	15	13.33	0.1637	0.0576	0.190	0.028	0.022	0.028
3,3'-DIMETHYLBENZIDINE	1	41	2.44	2.2555	3.9115	3.284	6.600	6.600	3.284
4,4'-DDD	4	52	7.69	0.0505	0.1844	0.094	0.410	0.017	0.094
4,4'-DDE	10	53	18.87	0.0583	0.1954	0.103	0.650	0.004	0.103
4,4'-DDT	15	52	28.85	0.0880	0.3551	0.171	0.510	0.001	0.171
4-CHLOROANILINE	13	41	31.71	1.1842	1.6765	1.625	7.400	0.098	1.625
ACENAPHTHENE	5	41	12.20	1.0934	1.9155	1.597	0.410	0.016	0.410
ACENAPHTHYLENE	6	41	14.63	1.0959	1.9154	1.600	0.160	0.042	0.160
ACETONE	1	52	1.92	0.1157	0.2890	0.183	0.320	0.320	0.183
ALDRIN (d)	3	53	5.66	0.0765	0.2611	0.137	1.100	0.130	0.137
ALPHA-BHC	7	53	13.21	0.0659	0.2377	0.121	1.200	0.001	0.121
ALPHA-CHLORDANE	6	52	11.54	0.0416	0.1753	0.083	0.077	0.002	0.077
AMMONIA AS N	5	26	19.23	0.9529	1.2825	1.382	5.200	1.100	1.382
ANTHRACENE	14	41	34.15	1.0405	1.9344	1.549	0.320	0.031	0.320
ANTIMONY	7	30	23.33	1.8440	7.5631	4.190	41.800	0.860	4.190
ARSENIC	34	34	100.00	7.6044	4.4865	8.910	16.200	0.550	8.910
BARIUM	44	44	100.00	90.2682	195.0021	139.774	1270.000	7.300	139.774
BENZENE	1	52	1.92	0.0414	0.0617	0.056	0.034	0.034	0.034
BENZO(A)ANTHRACENE	22	41	53.66	0.8341	1.1339	1.132	1.600	0.066	1.132
BENZO(A)PYRENE	20	41	48.78	0.9667	1.3988	1.335	1.700	0.025	1.335
BENZO(B)FLUORANTHENE	21	41	51.22	1.0710	1.2517	1.400	2.800	0.042	1.400
BENZO(G,H,I)PERYLENE	15	41	36.59	1.1703	1.8912	1.668	1.200	0.064	1.200
BENZO(K)FLUORANTHENE	19	41	46.34	1.0503	1.3890	1.416	3.600	0.062	1.416
BERYLLIUM (d)	43	44	97.73	0.6759	0.3732	0.771	2.000	0.230	0.771
BETA-BHC	2	52	3.85	0.0391	0.1754	0.080	0.010	0.009	0.010
BICARBONATE ALKALINITY	17	26	65.38	223.1346	222.0209	297.504	940.000	97.000	297.504
BIS(2-CHLOROETHYL)ETHER	2	41	4.88	1.0978	1.9121	1.601	0.430	0.330	0.430

Warwick Area - Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) ^c
BIS(2-ETHYLHEXYL)PHTHALATE	18	41	43.90	11.8848	36.3537	21.446	160.000	0.100	21.446
BUTYLBENZYLPHthalATE	7	41	17.07	1.1025	1.9132	1.606	0.780	0.046	0.780
CADMIUM	17	38	44.74	1.5504	2.2483	2.169	7.600	0.280	2.169
CALCIUM	26	26	100.00	1431.7692	795.3611	1698.188	3730.000	363.000	1698.188
CATION EXCHANGE CAPACITY	26	26	100.00	6.9731	4.9163	8.620	18.000	1.400	8.620
CHLORIDE	13	26	50.00	70.0000	85.6559	98.692	300.000	19.000	98.692
CHLOROBENZENE	12	52	23.08	10.0627	70.6917	26.571	510.000	0.006	26.571
CHLOROBENZILATE	1	55	1.82	0.8584	1.7274	1.251	0.046	0.046	0.046
CHLOROFORM	1	52	1.92	0.0416	0.0617	0.056	0.048	0.048	0.048
CHROMIUM	44	44	100.00	61.3545	114.0027	90.297	478.000	1.300	90.297
CHRYSENE	21	41	51.22	0.9178	1.1717	1.226	2.300	0.061	1.226
COBALT	34	34	100.00	4.2118	1.8383	4.747	7.800	1.000	4.747
COPPER	43	44	97.73	94.1318	300.5219	170.426	1960.000	1.800	170.426
CYANIDE	10	40	25.00	1.6928	2.3652	2.327	9.400	0.800	2.327
DELTA-BHC	4	53	7.55	0.0430	0.1764	0.084	0.260	0.002	0.084
DI-N-BUTYLPHthalATE	1	41	2.44	1.0965	1.9137	1.600	0.057	0.057	0.057
DI-N-OCTYLPHthalATE	10	41	24.39	4.1278	14.3585	7.904	89.000	0.040	7.904
DIBENZ(A,H)ANTHRACENE	6	41	14.63	1.0787	1.9240	1.585	0.130	0.045	0.130
DIBENZOFURAN	6	41	14.63	1.0720	1.9246	1.578	0.200	0.034	0.200
DIELDRIN (d)	6	52	11.54	0.0629	0.2179	0.114	0.910	0.002	0.114
DINOSEB	1	41	2.44	0.0740	0.0850	0.096	0.072	0.072	0.072
DIPHENYLAMINE	1	41	2.44	1.1078	1.9091	1.610	0.140	0.140	0.140
DISULFOTON	3	40	7.50	0.0695	0.0633	0.087	0.008	0.006	0.008
ENDOSULFAN I	4	52	7.69	0.0392	0.1754	0.080	0.018	0.002	0.018
ENDOSULFAN II	1	52	1.92	0.1071	0.5224	0.229	0.018	0.018	0.018
ENDOSULFAN SULFATE	4	52	7.69	0.1650	0.8343	0.360	0.290	0.007	0.290
ENDRIN	6	52	11.54	0.0565	0.1948	0.102	0.540	0.003	0.102
ENDRIN ALDEHYDE	6	52	11.54	0.1491	0.5911	0.287	3.500	0.002	0.287
ETHYL PARATHION	2	40	5.00	0.0344	0.0180	0.039	0.006	0.006	0.006
ETHYLBENZENE	1	52	1.92	0.0412	0.0617	0.056	0.027	0.027	0.027
FLUORANTHENE	24	41	58.54	1.0593	1.0647	1.339	3.700	0.038	1.339
FLUORENE	10	41	24.39	1.0714	1.9245	1.578	0.540	0.035	0.540
GAMMA-BHC	2	52	3.85	0.0388	0.1755	0.080	0.002	0.001	0.002
GAMMA-CHLORDANE	13	52	25.00	0.0594	0.1906	0.104	0.500	0.002	0.104
HEPTACHLOR	1	52	1.92	0.0388	0.1755	0.080	0.003	0.003	0.003
HEPTACHLOR EPOXIDE (d)	8	52	15.38	0.0695	0.2396	0.125	1.200	0.002	0.125
INDENO(1,2,3-CD)PYRENE	13	41	31.71	1.1297	1.9040	1.630	0.860	0.070	0.860
IRON	26	26	100.00	12215.0000	3693.0976	13452.063	20000.000	4610.000	13452.063
ISODRIN	2	52	3.85	0.0606	0.2136	0.110	0.850	0.007	0.110
KEPONE	1	52	1.92	0.1186	0.3691	0.205	0.260	0.260	0.205
LEAD	33	34	97.06	69.0103	96.0971	96.978	428.000	2.800	96.978
M&P-XYLENE	12	52	23.08	0.2441	1.5211	0.599	11.000	0.006	0.599

Warwick Area - Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) ^c
MAGNESIUM	26	26	100.00	1691.5000	1016.0961	2031.858	5360.000	290.000	2031.858
MANGANESE	26	26	100.00	228.1308	75.8917	253.552	416.000	83.400	253.552
MERCURY	20	38	52.63	0.2373	0.3122	0.323	1.300	0.090	0.323
METHOXYCHLOR (d)	22	54	40.74	105.8591	408.0589	199.371	2200.000	0.019	199.371
METHYLENE CHLORIDE	10	52	19.23	0.1796	0.3603	0.264	1.300	0.005	0.264
N-NITROSO-DI-N-PROPYLAMINE	1	41	2.44	1.0730	1.9254	1.579	0.170	0.170	0.170
NAPHTHALENE	21	41	51.22	1.6720	5.0374	2.997	32.000	0.036	2.997
NICKEL	41	44	93.18	13.7636	29.4651	21.244	199.000	2.200	21.244
NITRATE-NITRITE AS N	22	26	84.62	1.5358	1.4892	2.035	6.400	0.340	2.035
NITROBENZENE	2	41	4.88	1.1717	1.9237	1.678	2.900	0.480	1.678
O-XYLENE	5	52	9.62	0.1605	0.9252	0.377	6.700	0.017	0.377
OCDD	2	8	25.00	0.0007	0.0013	0.002	0.004	0.000	0.002
ORTHOPHOSPHATE	21	26	80.77	4.4146	6.6737	6.650	29.000	0.360	6.650
PCB-1248 (d)	3	54	5.56	4.4661	22.6419	9.655	160.000	8.100	9.655
PCB-1254 (d)	17	52	32.69	1.8958	6.2158	3.347	36.000	0.029	3.347
PERCENT MOISTURE	31	31	100.00	12.8000	8.6040	15.422	29.000	0.000	15.422
PH	16	16	100.00	6.1938	0.4795	6.404	7.100	5.600	6.404
PHENACETIN	1	41	2.44	1.1195	1.9065	1.621	1.000	1.000	1.000
PHENANTHRENE	25	41	60.98	0.7915	0.8232	1.008	2.300	0.033	1.008
PHENOL	4	41	9.76	1.0668	1.7734	1.533	5.000	0.350	1.533
PHORATE	1	40	2.50	0.0352	0.0173	0.040	0.010	0.010	0.010
POTASSIUM	26	26	100.00	755.5769	838.5637	1036.468	4630.000	331.000	1036.468
PROPAZINE	1	26	3.85	8.0269	10.8371	11.657	24.000	24.000	11.657
PYRENE	25	41	60.98	1.2800	1.3335	1.631	6.400	0.053	1.631
SAFROLE	4	41	9.76	1.9183	4.6142	3.132	28.000	0.700	3.132
SODIUM	16	26	61.54	119.6827	60.6326	139.993	217.000	118.000	139.993
SULFATE	11	26	42.31	124.9231	196.8096	190.848	890.000	13.000	190.848
SULFIDE	5	26	19.23	89.4423	328.9961	199.645	1700.000	35.000	199.645
SULFOTEPP	1	41	2.44	0.0221	0.0108	0.025	0.004	0.004	0.004
TETRACHLOROETHENE	14	52	26.92	0.1227	0.4336	0.224	2.400	0.007	0.224
THIONAZIN	1	40	2.50	0.1788	0.1301	0.214	0.006	0.006	0.006
TIN	6	38	15.79	6.7145	7.1763	8.690	37.800	14.700	8.690
TINUVIN 327	8	26	30.77	6.1988	8.4144	9.017	18.000	0.570	9.017
TINUVIN 328	1	5	20.00	0.2600	0.1623	0.415	0.550	0.550	0.415
TOLUENE	23	52	44.23	2.0701	13.8508	5.305	100.000	0.007	5.305
TOTAL ALKALINITY	17	26	65.38	223.1346	222.0209	297.504	940.000	97.000	297.504
TOTAL ORGANIC CARBON	26	26	100.00	11732.0000	11172.9005	15474.546	28000.000	240.000	15474.546
TRICHLOROETHENE	2	52	3.85	0.0428	0.0628	0.057	0.130	0.041	0.057
VANADIUM	26	34	76.47	9.5941	5.0737	11.071	22.000	1.700	11.071
ZINC	41	44	93.18	2000.6148	4000.9642	3016.350	16100.000	18.300	3016.350

Warwick Area - Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) ^c
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- a. Arithmetic mean concentration.
- b. 95th percentile upper confidence limit of the arithmetic mean concentration .
- c. Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
- d. Shading indicates that the chemical was selected as a chemical of potential concern (COPC).

Background - Surface Soil

TABLE A1-5
ANALYTICAL SUMMARY OF DETECTED COMPOUNDS
BACKGROUND
SURFACE SOILS

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)*	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)
1,2,3,4,6,7,8-HPCDF	1	4	25.00	4.85E-05	4.23E-05	9.83E-05	1.10E-04	1.10E-04
2-BUTANONE	1	12	8.33	0.087	0.057	0.117	0.170	0.170
2-METHYLNAPHTHALENE	2	12	16.67	0.664	1.220	1.297	4.500	0.570
4,4'-DDD	4	12	33.33	0.093	0.317	0.257	1.100	0.001
4,4'-DDE	6	12	50.00	0.105	0.247	0.233	0.810	0.001
4,4'-DDT	6	12	50.00	0.783	2.682	2.173	9.300	0.004
ACENAPHTHENE	5	12	41.67	0.690	1.501	1.469	5.400	0.031
ACENAPHTHYLENE	4	12	33.33	0.296	0.200	0.400	0.610	0.044
ALPHA-CHLORDANE	1	12	8.33	0.001	0.001	0.002	0.003	0.003
ANTHRACENE	7	12	58.33	2.166	5.735	5.139	20.000	0.041
ARSENIC	11	12	91.67	10.847	10.128	16.098	36.900	2.700
BARIUM	12	12	100.00	44.033	73.765	82.278	275.000	8.700
BENZENE	1	12	8.33	0.039	0.028	0.054	0.043	0.043
BENZO(A)ANTHRACENE	7	12	58.33	3.198	8.016	7.354	28.000	0.280
BENZO(A)PYRENE	8	12	66.67	2.566	6.266	5.814	22.000	0.130
BENZO(B)FLUORANTHENE	9	12	75.00	4.160	10.273	9.486	36.000	0.026
BENZO(G,H,I)PERYLENE	8	12	66.67	1.570	3.429	3.348	12.000	0.080
BENZO(K)FLUORANTHENE	8	12	66.67	4.700	12.292	11.073	43.000	0.079
BERYLLIUM	12	12	100.00	0.487	0.238	0.610	0.980	0.160
BETA-BHC	1	12	8.33	0.023	0.075	0.061	0.260	0.260
BIS(2-ETHYLHEXYL)PHTHALATE	2	11	18.18	0.367	0.321	0.542	0.120	0.087
BUTYLBENZYLPHTHALATE	1	11	9.09	0.303	0.191	0.407	0.050	0.050
CADMIUM	2	8	25.00	0.333	0.208	0.473	0.780	0.520
CALCIUM	8	8	100.00	926.375	322.369	1142.357	1440.000	560.000
CHLOROFORM	1	12	8.33	0.039	0.028	0.053	0.032	0.032
CHROMIUM	12	12	100.00	10.292	4.546	12.649	20.000	4.800
CHRYSENE	9	12	75.00	3.445	8.534	7.869	30.000	0.140
COBALT	12	12	100.00	3.467	1.690	4.343	7.000	1.800
COPPER	12	12	100.00	10.425	8.809	14.992	32.400	3.700
CYANIDE	1	8	12.50	0.964	0.909	1.573	3.000	3.000
DIBENZ(A,H)ANTHRACENE	3	12	25.00	0.612	1.010	1.135	3.700	0.120
DIBENZOFURAN	4	12	33.33	1.116	2.732	2.532	9.700	0.043
DIELDRIN	2	12	16.67	0.002	0.002	0.003	0.004	0.001
DINOSEB	1	12	8.33	0.063	0.085	0.107	0.003	0.003

Background - Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)
DISULFOTON	1	12	8.33	0.044	0.026	0.057	0.002	0.002
ENDOSULFAN SULFATE	1	12	8.33	0.003	0.002	0.004	0.006	0.006
ENDRIN	1	12	8.33	0.001	0.002	0.003	0.000	0.000
ENDRIN ALDEHYDE	1	12	8.33	0.002	0.002	0.003	0.001	0.001
ETHYL PARATHION	2	12	16.67	0.018	0.012	0.024	0.004	0.003
FLUORANTHENE	12	12	100.00	6.592	16.337	15.062	57.000	0.043
FLUORENE	5	12	41.67	1.105	2.651	2.479	9.400	0.053
GAMMA-CHLORDANE	1	12	8.33	0.001	0.002	0.002	0.005	0.005
HPCDF	1	4	25.00	0.000	0.000	0.000	0.000	0.000
INDENO(1,2,3-CD)PYRENE	7	12	58.33	1.830	4.029	3.919	14.000	0.230
IRON	8	8	100.00	12772.500	6867.927	17373.899	28300.000	7240.000
ISODRIN	1	12	8.33	0.002	0.002	0.003	0.003	0.003
KEPONE	2	12	16.67	0.022	0.052	0.049	0.180	0.055
LEAD	12	12	100.00	98.025	138.477	169.820	471.000	11.800
MAGNESIUM	8	8	100.00	1392.375	653.059	1829.914	2450.000	683.000
MANGANESE	8	8	100.00	202.525	145.874	300.258	476.000	53.200
MERCURY	4	12	33.33	0.133	0.226	0.250	0.810	0.060
METHYL PARATHION	4	12	33.33	0.005	0.003	0.007	0.005	0.003
METHYLENE CHLORIDE	5	12	41.67	0.295	0.665	0.639	0.120	0.010
NAPHTHALENE	4	12	33.33	0.861	2.042	1.920	7.300	0.023
NICKEL	8	12	66.67	5.608	3.277	7.307	13.300	3.200
OCDD	3	4	75.00	0.000	0.000	0.001	0.001	0.000
OCDF	1	4	25.00	0.000	0.000	0.000	0.000	0.000
P-PHENYLENEDIAMINE	1	11	9.09	2.041	1.545	2.885	5.900	5.900
PERCENT MOISTURE	12	12	100.00	12.317	6.833	15.859	22.000	2.000
PHENANTHRENE	11	12	91.67	7.210	19.742	17.446	69.000	0.052
POTASSIUM	8	8	100.00	504.250	144.534	601.086	786.000	349.000
PYRENE	12	12	100.00	6.284	16.002	14.580	56.000	0.038
SAFROLE	1	11	9.09	0.301	0.193	0.406	0.042	0.042
SELENIUM	3	12	25.00	0.350	0.284	0.497	1.100	0.490
SILVER	1	12	8.33	0.365	0.136	0.436	0.310	0.310
SODIUM	4	8	50.00	124.000	87.648	182.723	230.000	182.000
TETRACHLOROETHENE	1	12	8.33	0.042	0.028	0.056	0.011	0.011
THALLIUM	2	12	16.67	0.181	0.080	0.222	0.170	0.100
TIN	2	8	25.00	28.331	44.008	57.816	102.000	97.200
TOLUENE	4	12	33.33	0.140	0.337	0.315	1.200	0.027
VANADIUM	12	12	100.00	16.158	5.593	19.058	27.300	8.500
ZINC	12	12	100.00	46.442	56.900	75.942	219.000	13.300

Background - Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)
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a. Arithmetic mean concentration.

b. 95th percentile upper confidence limit of the arithmetic mean concentration .

Background - Combined Soils

TABLE A1-6
ANALYTICAL SUMMARY OF DETECTED COMPOUNDS
BACKGROUND
COMBINED SOILS

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)
1,2,3,4,6,7,8-HPCDF	1	9	0.111	3.81E-05	2.97E-05	5.65E-05	1.10E-04	1.10E-04	5.65E-05
2-BUTANONE	1	17	0.059	0.066	0.058	0.091	0.170	0.170	0.091
2-METHYLNAPHTHALENE	2	17	0.118	0.525	1.036	0.963	4.500	0.570	0.963
4,4'-DDD	4	17	0.235	0.066	0.266	0.179	1.100	0.001	0.179
4,4'-DDE	6	17	0.353	0.075	0.210	0.164	0.810	0.001	0.164
4,4'-DDT	7	17	0.412	0.553	2.254	1.508	9.300	0.004	1.508
7,12-DIMETHYLBENZ(A)ANTHRACENE	1	16	0.063	0.929	1.116	1.418	0.160	0.160	0.160
ACENAPHTHENE	5	17	0.294	0.543	1.267	1.080	5.400	0.031	1.080
ACENAPHTHYLENE	4	17	0.235	0.265	0.173	0.338	0.610	0.044	0.338
ALPHA-CHLORDANE	1	17	0.059	0.001	0.001	0.002	0.003	0.003	0.002
ANTHRACENE	8	17	0.471	1.575	4.848	3.628	20.000	0.036	3.628
ARSENIC	14	17	0.824	8.036	9.562	12.085	36.900	0.740	12.085
BARIUM	17	17	1.000	35.124	62.876	61.750	275.000	6.400	61.750
BENZENE	1	17	0.059	0.029	0.029	0.041	0.043	0.043	0.041
BENZO(A)ANTHRACENE	9	17	0.529	2.293	6.802	5.173	28.000	0.088	5.173
BENZO(A)PYRENE	10	17	0.588	1.847	5.321	4.100	22.000	0.086	4.100
BENZO(B)FLUORANTHENE	11	17	0.647	2.976	8.726	6.671	36.000	0.026	6.671
BENZO(G,H,I)PERYLENE	9	17	0.529	1.156	2.919	2.393	12.000	0.045	2.393
BENZO(K)FLUORANTHENE	10	17	0.588	3.348	10.418	7.760	43.000	0.051	7.760
BERYLLIUM	16	17	0.941	0.490	0.262	0.601	0.980	0.160	0.601
BETA-BHC	1	17	0.059	0.016	0.063	0.043	0.260	0.260	0.043
BIS(2-ETHYLHEXYL)PHTHALATE	2	16	0.125	0.312	0.276	0.432	0.120	0.087	0.120
BUTYLBENZYLPHTHALATE	1	16	0.063	0.268	0.165	0.340	0.050	0.050	0.050
CADMIUM	2	9	0.222	0.313	0.204	0.440	0.780	0.520	0.440
CALCIUM	8	8	1.000	926.375	322.369	1142.357	1440.000	560.000	1142.357
CHLOROFORM	2	17	0.118	0.028	0.028	0.040	0.032	0.007	0.032
CHROMIUM	17	17	1.000	8.788	4.591	10.733	20.000	3.200	10.733
CHRYSENE	11	17	0.647	2.482	7.241	5.548	30.000	0.088	5.548
COBALT	17	17	1.000	3.182	1.559	3.842	7.000	1.700	3.842
COPPER	17	17	1.000	9.082	7.632	12.314	32.400	3.700	12.314
CYANIDE	1	13	0.077	0.978	0.700	1.324	3.000	3.000	1.324
DIBENZ(A,H)ANTHRACENE	3	17	0.176	0.466	0.869	0.834	3.700	0.120	0.834
DIBENZOFURAN	4	17	0.235	0.844	2.306	1.820	9.700	0.043	1.820
DIELDRIN	2	17	0.118	0.002	0.002	0.003	0.004	0.001	0.003
DINOSEB	1	16	0.063	0.096	0.093	0.137	0.003	0.003	0.003
DISULFOTON	1	16	0.063	0.052	0.026	0.064	0.002	0.002	0.002

Background - Combined Soils

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION	MEAN CONC. (MG/KG) ^a	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) ^b	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)
ENDOSULFAN SULFATE	1	17	0.059	0.003	0.002	0.004	0.006	0.006	0.004
ENDRIN	1	17	0.059	0.002	0.002	0.002	0.000	0.000	0.000
ENDRIN ALDEHYDE	1	17	0.059	0.002	0.002	0.003	0.001	0.001	0.001
ETHYL PARATHION	2	16	0.125	0.019	0.010	0.023	0.004	0.003	0.004
FLUORANTHENE	16	17	0.941	4.697	13.880	10.575	57.000	0.043	10.575
FLUORENE	5	17	0.294	0.836	2.239	1.784	9.400	0.053	1.784
GAMMA-CHLORDANE	1	17	0.059	0.001	0.001	0.002	0.005	0.005	0.002
HPCDD	1	9	0.111	0.000	0.000	0.000	0.000	0.000	0.000
HPCDF	1	9	0.111	0.000	0.000	0.000	0.000	0.000	0.000
INDENO(1,2,3-CD)PYRENE	9	17	0.529	1.320	3.438	2.776	14.000	0.050	2.776
IRON	8	8	1.000	12772.500	6867.927	17373.899	28300.000	7240.000	17373.899
ISODRIN	1	17	0.059	0.002	0.002	0.003	0.003	0.003	0.003
KEPONE	2	17	0.118	0.019	0.044	0.037	0.180	0.055	0.037
LEAD	17	17	1.000	72.235	122.088	123.936	471.000	2.900	123.936
MAGNESIUM	8	8	1.000	1392.375	653.059	1829.914	2450.000	683.000	1829.914
MANGANESE	8	8	1.000	202.525	145.874	300.258	476.000	53.200	300.258
MERCURY	4	17	0.235	0.107	0.192	0.188	0.810	0.060	0.188
METHOXYCHLOR	2	17	0.118	0.058	0.199	0.142	0.830	0.034	0.142
METHYL PARATHION	4	16	0.250	0.007	0.003	0.008	0.005	0.003	0.005
METHYLENE CHLORIDE	7	17	0.412	0.210	0.567	0.450	0.120	0.010	0.120
NAPHTHALENE	4	17	0.235	0.664	1.722	1.393	7.300	0.023	1.393
NICKEL	13	17	0.765	5.124	2.925	6.362	13.300	2.600	6.362
OCDD	4	9	0.444	0.000	0.000	0.001	0.001	0.000	0.001
OCDF	1	9	0.111	0.000	0.000	0.000	0.000	0.000	0.000
P-PHENYLENEDIAMINE	1	16	0.063	1.706	1.362	2.303	5.900	5.900	2.303
PERCENT MOISTURE	15	15	1.000	12.253	6.480	15.200	22.000	2.000	15.200
PHENANTHRENE	13	17	0.765	5.141	16.700	12.213	69.000	0.052	12.213
POTASSIUM	8	8	1.000	504.250	144.534	601.086	786.000	349.000	601.086
PYRENE	16	17	0.941	4.482	13.576	10.231	56.000	0.037	10.231
SAFROLE	1	16	0.063	0.266	0.166	0.339	0.042	0.042	0.042
SELENIUM	3	17	0.176	0.349	0.290	0.472	1.100	0.490	0.472
SILVER	2	17	0.118	0.313	0.148	0.375	0.350	0.310	0.350
SODIUM	4	8	0.500	124.000	87.648	182.723	230.000	182.000	182.723
TETRACHLOROETHENE	1	17	0.059	0.031	0.029	0.043	0.011	0.011	0.011
THALLIUM	3	17	0.176	0.170	0.099	0.212	0.210	0.100	0.210
TIN	2	9	0.222	30.961	41.915	56.948	102.000	97.200	56.948
TOLUENE	4	17	0.235	0.100	0.287	0.221	1.200	0.027	0.221
VANADIUM	17	17	1.000	13.412	6.559	16.189	27.300	4.300	16.189
ZINC	17	17	1.000	40.241	48.538	60.795	219.000	12.700	60.795

a. Arithmetic mean concentration.

b. 95th percentile upper confidence limit of the arithmetic mean concentration .

Attachment 2

Chemicals of Potential Concern Selection Process and Results

A2-1.0 Introduction

Chemicals of potential concern (COPC) for this Risk Assessment were selected separately for the Production and Warwick Areas. The selection process was based on USEPA guidance (USEPA, 1989a), previous discussions with USEPA Region I and an evaluation of the analytical data. The purpose of this selection process is to limit the Risk Assessment to those chemicals which represent the dominant human health risks. The data evaluated include Phase I (Rounds 1 and 2) and Phase II (Rounds 1 and 2) RCRA Facility Investigation soils data. During COPC selection, no Phase I data were used for polychlorinated dibenzodioxins and dibenzofurans (PCDD/Fs) due to QA/QC validation issues.

A2-2.0 Methodology

Only those chemicals which were detected in at least 5 percent of the surface soil samples from a Site area were included in the selection process. Chemicals that are infrequently detected may be artifacts in the data due to sampling errors, analytical errors, or other problems.

The first step in the selection of COPC was the evaluation of background chemical concentrations. Background levels were evaluated for inorganic chemicals and PAHs. Inorganic chemicals are naturally present in soil. PAHs are ubiquitous in surface soil due to emissions from non-site-related combustion sources such as automobiles, industrial burners, and charcoal grills. Inorganic chemicals and PAH compounds were eliminated from further consideration if the mean sample concentration was less than the 95th percentile upper confidence limit (UCL) of the mean of background concentrations. Evaluation of background is discussed in detail in Section A3.0 of the Risk Assessment.

A quantitative concentration/toxicity relative ranking system was used after completing the background screen to rank the chemicals detected in surface soil at each area according to their potential contribution to human health risk at the Site (USEPA, 1989). The objective of this ranking procedure is to identify the chemicals that are most likely to contribute significantly to risks at the Site. The ranking procedure has three steps. First a ranking factor was calculated for

each chemical. This ranking factor is based on the soil concentrations detected in the Production and Warwick Areas, and toxicity of the chemical. The ranking factor for each chemical in each medium was calculated as shown below:

$$R_i = (C_i)(T_i)$$

Where:

- R_i = Ranking factor for chemical i .
- C_i = Concentration of chemical i .
- T_i = Toxicity criterion of chemical i (either the CSF or 1/RfD of chemical i see "Toxicity Assessment" for description).

The concentration used is the 95th percentile UCL of the mean of the surface soil sample concentrations. The Integrated Risk Information System (IRIS) on-line database, Health Effect Assessment Summary Tables (USEPA, 1994), Appendix X of the 1993 Revised Cranston RFI Interim Report and Phase 2 Work Plan (Ciba, 1993), and information from Ciba were the sources for toxicity criteria.

Next, a total score was calculated by summing the chemical-specific ranking factor values:

$$TS = R_i + R_{ii} + R_{iii} + \dots + R_n$$

Where:

- TS = Total score for all chemicals.
- R_i = Ranking factor for chemical i .

Finally the relative ranking score of each chemical was determined by dividing its ranking factor by the total score:

$$RRS_i = R_i/TS$$

Where:

- RRS_{*i*} = Relative ranking score of chemical i . (R_i and TS are as described above.)

Separate RRS values were calculated for cancer and noncancer effects. The results of the relative ranking system were used to select COPC by sequentially selecting noncarcinogens beginning

with the chemical having the highest relative ranking score until a cumulative relative ranking score of 0.9 was reached. A minimum of two chemicals was selected for each area. This was repeated to identify the carcinogens with the highest contribution to the TS value for carcinogenicity. To ensure that all chemicals which contribute significantly to risk at the Site were included in the Risk Assessment, the following iterative evaluation process was performed after completion of the risk analyses during the risk characterization process.

- For noncarcinogens - if a total hazard index of 1 is exceeded in the risk calculations for the selected COPC, noncarcinogens with the highest remaining relative risk scores are added sequentially to the list of COPC until two additional compounds with estimated hazard quotients of less than 0.5 are included on the list.
- For carcinogens - if the total incremental lifetime cancer risk (ILCR) associated with the selected COPC exceeds 1×10^{-4} , carcinogens with the highest remaining relative risk scores are added sequentially to the list of COPC until two additional compounds with estimated ILCR values of less than 1×10^{-5} are included on the list.

A2-3.0 Results

Surface soil COPC selected for the Production and Warwick areas are as follows:

PRODUCTION AREA

Noncarcinogens

PCB 1248

PCB 1254

Carcinogens

PCB 1260

gamma-Chlordane

WARWICK AREA

Noncarcinogens

PCB 1248

PCB 1254

2-Nitroaniline

Methoxychlor

Carcinogens

Aldrin

Beryllium

Dieldrin

Heptachlor epoxide

Summaries of the data and COPC selection are provided in this attachment in Tables A2-1 through A2-3 for the Production Area and Tables A2-4 through A2-6 for the Warwick Area. These tables provide the following information for surface soil data at each area:

- Frequency of detection;
- Mean concentration;
- Reasonable Maximum Exposure (RME) concentration;
- Background concentration (for inorganics and PAHs);
- Determination of whether the mean concentration exceeds background levels for inorganics and PAHs;
- Cancer and/or noncancer toxicity criteria; and
- A relative ranking score based on the above information.

The results of the COPC selection process were reviewed to ensure that the COPC included compounds known to be previously used or produced at the Site and those identified as concerns during previous discussions with USEPA, such as polychlorinated biphenyls (PCBs).

TABLE A2-1
Production Area--Surface Soil

Ranking Process for Chemicals of Potential Concern Selection

Ciba-Geigy Corporation, Cranston Rhode Island Site

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ⁻¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
ORGANICS											
ACENAPHTHENE	10	41	0.24	1.24	0.21	1.47	No	NA	NA	NA	NA
ACENAPHTHYLENE	5	41	0.12	1.26	0.18	0.40	No	NA	NA	NA	NA
ANTHRACENE	24	41	0.59	0.88	1.29	5.14	No	NA	NA	NA	NA
BENZO(A)ANTHRACENE	28	41	0.68	1.14	1.52	7.35	No	NA	NA	NA	NA
BENZO(A)PYRENE	27	41	0.66	1.29	1.69	5.81	No	NA	NA	NA	NA
BENZO(B)FLUORANTHENE	30	41	0.73	1.57	1.98	9.49	No	NA	NA	NA	NA
BENZO(G,H,I)PERYLENE	21	41	0.51	1.35	1.81	3.35	No	NA	NA	NA	NA
BENZO(K)FLUORANTHENE	27	41	0.66	1.50	1.95	11.07	No	NA	NA	NA	NA
BIS(2-ETHYLHEXYL)PHTHALATE	16	41	0.39	1.09	1.49	--	NA	1.4E-2	--	0.000	--
BUTYLBENZYLPHthalATE	13	41	0.32	1.83	3.22	--	NA	--	2.0E-1	--	0.000
GAMMA-CHLORDANE	7	43	0.16	0.07	0.13	--	NA	1.3E+0	6.5E-5	0.004	0.000
4-CHLOROANILINE	5	41	0.12	1.32	0.64	--	NA	--	4.0E-3	--	0.000
CHLOROBENZENE	7	40	0.18	0.20	0.28	--	NA	--	2.0E-2	--	0.000
CHRYSENE	28	41	0.68	1.24	1.62	7.87	No	NA	NA	NA	NA
DI-N-BUTYLPHthalATE	8	41	0.20	1.00	1.30	--	NA	--	1.0E-1	--	0.000
DIBENZ(A,H)ANTHRACENE	10	41	0.24	1.23	0.68	1.14	No	NA	NA	NA	NA
DIBENZOFURAN	12	41	0.29	1.20	0.13	--	NA	--	1.0E-2	--	0.000
DINOSEB	3	40	0.08	0.07	0.01	--	NA	--	1.0E-3	--	0.000
ETHYLBENZENE	10	40	0.25	1.29	3.41	--	NA	--	1.0E-1	--	0.000
FLUORANTHENE	33	41	0.80	1.60	2.05	15.06	No	NA	NA	NA	NA
FLUORENE	12	41	0.29	1.21	0.18	2.48	No	NA	NA	NA	NA
INDENO(1,2,3-CD)PYRENE	21	41	0.51	1.33	1.79	3.92	No	NA	NA	NA	NA

TABLE A2-1
Production Area--Surface Soil

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ⁻¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
IRGASAN DP-300	3	26	0.12	9.42	4.20	--	NA	--	3.0E-1	--	0.000
METHOXYCHLOR	7	43	0.16	0.35	0.55	--	NA	--	5.0E-3	--	0.000
METHYLENE CHLORIDE	6	40	0.15	0.27	0.01	--	NA	7.5E-3	6.0E-2	0.000	0.000
2-METHYLNAPHTHALENE	4	41	0.10	1.26	0.38	--	NA	--	--	--	--
3&4-METHYLPHENOL	5	16	0.31	0.18	0.25	--	NA	--	5.0E-2	--	0.000
NAPHTHALENE	14	41	0.34	0.83	0.68	1.92	No	NA	NA	NA	NA
2-NITROANILINE	8	41	0.20	6.25	0.89	--	NA	--	6.0E-5	--	0.003
NITROBENZENE	3	41	0.07	1.25	0.14	--	NA	--	5.0E-4	--	0.000
OCDD	1	5	0.20	0.00	0.00	--	NA	1.6E+2	--	0.002	--
ORTHOPHOSPHATE	20	25	0.80	5.15	8.13	--	NA	--	--	--	--
PCB-1248	38	98	0.39	53.50	130.49	--	NA	--	3.0E-5	--	0.811
PCB-1254	95	107	0.89	15.36	19.76	--	NA	--	2.0E-5	--	0.184
PCB-1260	7	52	0.13	4.65	6.10	--	NA	7.7E+0	--	0.994	--
PHENANTHRENE	28	41	0.68	1.15	1.54	17.45	No	NA	NA	NA	NA
PYRENE	32	41	0.78	1.82	2.30	14.58	No	NA	NA	NA	NA
TCDF	3	5	0.60	0.00	0.00	--	NA	1.6E+2	--	0.001	--
TINUVIN 328	3	3	1.00	4.53	5.90	--	NA	--	1.5E-2	--	0.000
TOLUENE	18	40	0.45	0.39	0.71	--	NA	--	2.0E-1	--	0.000
TRCDF	2	5	0.40	0.23	0.54	--	NA	--	1.0E-2	--	0.000
TRICHLOROFLUOROMETHANE	3	40	0.08	0.41	0.33	--	NA	--	3.0E-1	--	0.000
M&P-XYLENE	27	40	0.68	10.08	27.05	--	NA	--	2.0E+0	--	0.000
O-XYLENE	19	40	0.48	3.03	8.12	--	NA	--	2.0E+0	--	0.000
INORGANICS											
ARSENIC	29	32	0.91	9.01	15.43	16.10	No	NA	NA	NA	NA
BARIUM	31	31	1.00	46.54	56.20	82.28	No	NA	NA	NA	NA
BERYLLIUM	30	31	0.97	0.40	0.45	0.61	No	NA	NA	NA	NA

TABLE A2-1
Production Area--Surface Soil

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ⁻¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
CADMIUM	18	31	0.58	0.65	0.87	0.47	Yes	—	5.0E-4	—	0.000
CHROMIUM	29	31	0.94	11.13	13.66	12.65	No	NA	NA	NA	NA
COBALT	29	31	0.94	3.04	3.44	4.34	No	NA	NA	NA	NA
COPPER	29	31	0.94	17.62	22.71	14.99	Yes	—	3.7E-2	—	0.000
CYANIDE	11	34	0.32	1.32	1.98	1.57	No	NA	NA	NA	NA
IRON	26	26	1.00	10472.31	11821.61	17373.90	No	NA	NA	NA	NA
LEAD	29	31	0.94	54.31	79.46	169.82	No	NA	NA	NA	NA
MAGNESIUM	26	26	1.00	2114.92	2540.43	1829.91	Yes	—	—	—	—
MANGANESE	26	26	1.00	166.93	189.23	300.26	No	NA	NA	NA	NA
MERCURY	22	30	0.73	0.46	0.61	0.25	Yes	—	3.0E-4	—	0.000
NICKEL	28	31	0.90	7.77	9.52	7.31	Yes	—	2.0E-2	—	0.000
POTASSIUM	25	26	0.96	841.87	939.71	601.09	Yes	—	—	—	—
SODIUM	13	26	0.50	150.62	176.76	182.72	No	NA	NA	NA	NA
VANADIUM	28	31	0.90	14.98	20.76	19.06	No	NA	NA	NA	NA
ZINC	31	31	1.00	183.61	239.84	75.94	Yes	—	3.0E-1	—	0.000

¹Shading indicates compounds selected for the Risk Assessment.

²Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

³Selected concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected). If the 95% UCL is greater than the maximum detected concentration, the selected concentration is the maximum detected concentration.

⁴95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

⁵Exceeds background concentrations. Background concentrations were considered in the ranking of inorganics and PAHs. A background concentration of zero was assumed during the ranking process for all other organic compounds. A 'Yes' indicates that the mean sample concentration exceeds the background concentration; these compounds are carried through the ranking process. A 'No' indicates the mean sample concentration does not exceed the background concentration; these compounds were eliminated as COPCs and were not carried through the ranking process.

TABLE A2-1
Production Area--Surface Soil

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ⁻¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
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¹Cancer slope factor; NA - not applicable - no CSF is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

⁷Chronic reference dose; NA - not applicable - no RfD is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

⁶Relative ranking score--carcinogenic.

⁹Relative ranking score--noncarcinogenic.

TABLE A2-2
Production Area--Surface Soil

Ranking Process for Chemicals of Potential Concern Selection: Summary of Carcinogens
Ciba-Geigy Corporation, Cranston Rhode Island Site

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ⁻¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
ORGANICS											
PCB-1260	7	52	0.13	4.65	6.10	--	NA	7.7E+0	--	0.994	--
GAMMA-CHLORDANE	7	43	0.16	0.07	0.13	--	NA	1.3E+0	6.5E-5	0.004	0.000
OCDD	1	5	0.20	0.00	0.00	--	NA	1.6E+2	--	0.002	--
TCDF	3	5	0.60	0.00	0.00	--	NA	1.6E+2	--	0.001	--

¹Shading indicates compounds selected for the Risk Assessment.

²Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

³Selected concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected). If the 95% UCL is greater than the maximum detected concentration, the selected concentration is the maximum detected concentration.

⁴95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

⁵Exceeds background concentrations. Background concentrations were considered in the ranking of inorganics and PAHs. A background concentration of zero was assumed during the ranking process for all other organic compounds. A 'Yes' indicates that the mean sample concentration exceeds the background concentration; these compounds are carried through the ranking process. A 'No' indicates the mean sample concentration does not exceed the background concentration; these compounds were eliminated as COPCs and were not carried through the ranking process.

⁶Cancer slope factor; NA - not applicable - no CSF is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

⁷Chronic reference dose; NA - not applicable - no RfD is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

⁸Relative ranking score--carcinogenic.

⁹Relative ranking score--noncarcinogenic.

**TABLE A2-3
Production Area--Surface Soil**

Ranking Process for Chemicals of Potential Concern Selection: Summary of Noncarcinogens

Ciba-Geigy Corporation, Cranston Rhode Island Site

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ⁻¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
ORGANICS											
PCB-1248	38	98	0.39	53.50	130.49	—	NA	—	3.0E-5	—	0.811
PCB-1254	95	107	0.89	15.36	19.76	—	NA	—	2.0E-5	—	0.184
2-NITROANILINE	8	41	0.20	6.25	0.89	—	NA	—	6.0E-5	—	0.003

¹Shading indicates compounds selected for the Risk Assessment.

²Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

³Selected concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected). If the 95% UCL is greater than the maximum detected concentration, the selected concentration is the maximum detected concentration.

⁴95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

⁵Exceeds background concentrations. Background concentrations were considered in the ranking of inorganics and PAHs. A background concentration of zero was assumed during the ranking process for all other organic compounds. A 'Yes' indicates that the mean sample concentration exceeds the background concentration; these compounds are carried through the ranking process. A 'No' indicates the mean sample concentration does not exceed the background concentration; these compounds were eliminated as COPCs and were not carried through the ranking process.

⁶Cancer slope factor; NA - not applicable - no CSF is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

⁷Chronic reference dose; NA - not applicable - no RfD is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

⁸Relative ranking score—carcinogenic.

⁹Relative ranking score—noncarcinogenic.

TABLE A2-4
Warwick Area--Surface Soil

Ranking Process for Chemicals of Potential Concern Selection

Ciba-Geigy Corporation, Cranston Rhode Island Site

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ⁻¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
ORGANICS											
ACENAPHTHENE	3	31	0.10	1.37	0.16	1.47	No	NA	NA	NA	NA
ACENAPHTHYLENE	3	31	0.10	1.39	0.11	0.40	No	NA	NA	NA	NA
ALDRIN	3	33	0.09	0.11	0.21	—	NA	1.7E+1	—	0.278	—
ALPHA-BHC	6	33	0.18	0.09	0.18	—	NA	3.4E-1	—	0.005	—
ALPHA-CHLORDANE	3	32	0.09	0.05	0.08	—	NA	1.3E+0	6.0E-5	0.008	0.001
ANTHRACENE	10	31	0.32	1.31	0.32	5.14	No	NA	NA	NA	NA
BENZO(A)ANTHRACENE	15	31	0.48	0.97	1.36	7.35	No	NA	NA	NA	NA
BENZO(A)PYRENE	13	31	0.42	1.15	1.62	5.81	No	NA	NA	NA	NA
BENZO(B)FLUORANTHENE	14	31	0.45	1.23	1.65	9.49	No	NA	NA	NA	NA
BENZO(G,H,I)PERYLENE	9	31	0.29	1.45	1.20	3.35	No	NA	NA	NA	NA
BENZO(K)FLUORANTHENE	13	31	0.42	1.30	1.76	11.07	No	NA	NA	NA	NA
BETA-BHC	2	32	0.06	0.05	0.01	—	NA	6.3E+0	—	0.005	—
GAMMA-BHC	2	32	0.06	0.05	0.00	—	NA	—	3.0E-4	—	0.000
1,1-BIPHENYL	1	2	0.50	1.04	1.90	—	NA	—	5.0E-2	—	0.000
BIS(2-CHLOROETHYL)ETHER	2	31	0.06	1.38	0.43	—	NA	1.1E+0	—	0.037	—
BIS(2-ETHYLHEXYL)PHTHALATE	14	31	0.45	10.46	20.04	—	NA	1.4E-2	—	0.022	—
2-BUTANONE	2	32	0.06	0.10	0.12	—	NA	—	6.0E-1	—	0.000
BUTYLBENZYLPHTHALATE	6	31	0.19	1.39	0.78	—	NA	—	2.0E-1	—	0.000
GAMMA-CHLORDANE	9	32	0.28	0.08	0.15	—	NA	1.3E+0	6.5E-5	0.015	0.002
4-CHLOROANILINE	10	31	0.32	1.44	2.01	—	NA	—	4.0E-3	—	0.000
CHLOROBENZENE	9	32	0.28	0.41	0.67	—	NA	—	2.0E-2	—	0.000
CHRYSENE	14	31	0.45	1.08	1.47	7.87	No	NA	NA	NA	NA
4,4'-DDD	2	32	0.08	0.07	0.13	—	NA	2.4E-1	—	0.003	—
4,4'-DDE	6	33	0.18	0.08	0.15	—	NA	3.4E-1	—	0.004	—

TABLE A2-4
Warwick Area--Surface Soil

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
4,4'-DDT	9	32	0.28	0.12	0.25	—	NA	3.4E-1	—	0.007	—
DELTA-BHC	3	33	0.09	0.06	0.12	—	NA	—	3.0E-4	—	0.000
DI-N-OCTYLPHTHALATE	8	31	0.26	2.52	4.12	—	NA	—	2.0E-2	—	0.000
DIBENZ(A,H)ANTHRACENE	3	31	0.10	1.38	0.13	1.14	No	NA	NA	NA	NA
DIBENZOFURAN	3	31	0.10	1.36	0.20	—	NA	—	1.0E-2	—	0.000
1,4-DICHLOROBENZENE	2	31	0.06	1.39	0.04	—	NA	2.4E-2	—	0.000	—
DIELDRIN	5	32	0.16	0.08	0.16	—	NA	1.6E+1	5.0E-5	0.202	0.003
DISULFOTON	3	30	0.10	0.06	0.01	—	NA	—	4.0E-5	—	0.000
ENDOSULFAN I	3	32	0.09	0.05	0.02	—	NA	—	6.0E-3	—	0.000
ENDOSULFAN SULFATE	3	32	0.09	0.25	0.29	—	NA	—	6.0E-3	—	0.000
ENDRIN	6	32	0.19	0.07	0.14	—	NA	—	3.0E-4	—	0.000
ENDRIN ALDEHYDE	5	32	0.16	0.22	0.44	—	NA	—	3.0E-4	—	0.001
ETHYL PARATHION	2	30	0.07	0.04	0.01	—	NA	—	—	—	—
FLUORANTHENE	17	31	0.55	1.23	1.58	15.06	No	NA	NA	NA	NA
FLUORENE	7	31	0.23	1.34	0.23	2.48	No	NA	NA	NA	NA
HEPTACHLOR EPOXIDE	7	32	0.22	0.10	0.19	—	NA	9.1E+0	1.3E-5	0.137	0.014
INDENO(1,2,3-CD)PYRENE	8	31	0.26	1.41	0.86	3.92	No	NA	NA	NA	NA
ISODRIN	2	32	0.06	0.08	0.16	—	NA	—	3.0E-5	—	0.005
METHOXYCHLOR	13	34	0.38	110.39	231.88	—	NA	—	5.0E-3	—	0.045
METHYLENE CHLORIDE	3	32	0.09	0.24	0.03	—	NA	7.5E-3	6.0E-2	0.000	0.000
2-METHYLNAPHTHALENE	6	31	0.19	1.34	0.36	—	NA	—	—	—	—
3&4-METHYLPHENOL	1	6	0.17	0.15	0.02	—	NA	—	5.0E-2	—	0.000
NAPHTHALENE	16	31	0.52	1.13	1.58	1.92	No	NA	NA	NA	NA
2-NITROANILINE	2	31	0.06	7.15	7.00	—	NA	—	6.0E-5	—	0.114
NITROBENZENE	2	31	0.06	1.48	2.13	—	NA	—	5.0E-4	—	0.004
ORTHOPHOSPHATE	20	25	0.80	4.57	6.88	—	NA	—	—	—	—
PCB-1248	3	34	0.09	6.89	15.16	—	NA	—	3.0E-5	—	0.492
PCB-1254	15	32	0.47	2.86	5.19	—	NA	—	2.0E-5	—	0.253
PHENANTHRENE	17	31	0.55	0.87	1.13	17.45	No	NA	NA	NA	NA
PHENOL	3	31	0.10	1.18	0.89	—	NA	—	6.0E-1	—	0.000

TABLE A2-4
Warwick Area--Surface Soil

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
PYRENE	18	31	0.58	1.28	1.60	14.58	No	NA	NA	NA	NA
SAFROLE	4	31	0.13	2.46	4.05	—	NA	—	2.0E-2	—	0.000
TETRACHLOROETHENE	9	32	0.28	0.13	0.25	—	NA	5.2E-2	—	0.001	—
TINUVIN 327	8	25	0.32	6.34	9.27	—	NA	—	2.5E-3	—	0.004
TOLUENE	18	32	0.56	0.23	0.36	—	NA	—	2.0E-1	—	0.000
2,4,5-TP (SILVEX)	4	31	0.13	0.03	0.05	—	NA	—	8.0E-3	—	0.000
TRICHLOROETHENE	2	32	0.06	0.05	0.06	—	NA	1.1E-2	—	0.000	—
M&P-XYLENE	8	32	0.25	0.05	0.05	—	NA	—	2.0E+0	—	0.000
O-XYLENE	4	32	0.13	0.04	0.05	—	NA	—	2.0E+0	—	0.000
INORGANICS											
ANTIMONY	6	23	0.26	2.27	5.35	0.00	Yes	—	4.0E-4	—	0.013
ARSENIC	27	27	1.00	9.00	10.25	16.10	No	NA	NA	NA	NA
BARIUM	31	31	1.00	113.65	183.05	82.28	Yes	—	7.0E-2	—	0.003
BERYLLIUM	31	31	1.00	0.70	0.82	0.61	Yes	4.3E+0	5.0E-3	0.276	0.000
CADMIUM	15	31	0.48	1.56	2.22	0.47	Yes	—	5.0E-4	—	0.004
CHROMIUM	31	31	1.00	68.15	101.04	12.65	Yes	—	5.0E-3	—	0.020
COBALT	27	27	1.00	4.43	5.01	4.34	Yes	—	6.0E-2	—	0.000
COPPER	30	31	0.97	118.02	225.23	14.99	Yes	—	3.7E-2	—	0.006
CYANIDE	9	30	0.30	1.86	2.70	1.57	Yes	—	4.0E-2	—	0.000
IRON	25	25	1.00	12327.60	13601.76	17373.90	No	NA	NA	NA	NA
LEAD	26	27	0.96	84.01	117.72	169.82	No	NA	NA	NA	NA
MAGNESIUM	25	25	1.00	1713.56	2066.26	1829.91	No	NA	NA	NA	NA
MANGANESE	25	25	1.00	221.14	244.53	300.26	No	NA	NA	NA	NA
MERCURY	15	31	0.48	0.23	0.31	0.25	No	NA	NA	NA	NA
NICKEL	28	31	0.90	17.01	27.56	7.31	Yes	—	2.0E-2	—	0.001
POTASSIUM	25	25	1.00	767.44	1059.55	601.09	Yes	—	—	—	—
SODIUM	15	25	0.60	117.23	137.95	182.72	No	NA	NA	NA	NA
TIN	6	31	0.19	7.42	9.75	57.82	No	NA	NA	NA	NA

**TABLE A2-4
Warwick Area--Surface Soil**

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ⁻¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
VANADIUM	19	27	0.70	10.51	12.24	19.06	No	NA	NA	NA	NA
ZINC	28	31	0.90	2538.67	3911.97	75.94	Yes	—	3.0E-1	—	0.013

¹Shading indicates compounds selected for the Risk Assessment.

²Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

³Selected concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected). If the 95% UCL is greater than the maximum detected concentration, the selected concentration is the maximum detected concentration.

⁴95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

⁵Exceeds background concentrations. Background concentrations were considered in the ranking of inorganics and PAHs. A background concentration of zero was assumed during the ranking process for all other organic compounds. A 'Yes' indicates that the mean sample concentration exceeds the background concentration; these compounds are carried through the ranking process. A 'No' indicates the mean sample concentration does not exceed the background concentration; these compounds were eliminated as COPCs and were not carried through the ranking process.

⁶Cancer slope factor; NA - not applicable - no CSF is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

⁷Chronic reference dose; NA - not applicable - no RfD is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

⁸Relative ranking score—carcinogenic.

⁹Relative ranking score—noncarcinogenic.

TABLE A2-5
Warwick Area--Surface Soil

Ranking Process for Chemicals of Potential Concern Selection: Summary of Carcinogens

Ciba-Geigy Corporation, Cranston Rhode Island Site

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ⁻¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
ORGANICS											
ALDRIN	3	33	0.09	0.11	0.21	—	NA	1.7E+1	—	0.278	—
BERYLLIUM	31	31	1.00	0.70	0.82	0.61	Yes	4.3E+0	5.0E-3	0.276	0.000
DIELDRIN	5	32	0.16	0.08	0.16	—	NA	1.6E+1	5.0E-5	0.202	0.003
HEPTACHLOR EPOXIDE	7	32	0.22	0.10	0.19	—	NA	9.1E+0	1.3E-5	0.137	0.014
BIS(2-CHLOROETHYL)ETHER	2	31	0.06	1.38	0.43	—	NA	1.1E+0	—	0.037	—
BIS(2-ETHYLHEXYL)PHTHALATE	14	31	0.45	10.46	20.04	—	NA	1.4E-2	—	0.022	—
GAMMA-CHLORDANE	9	32	0.28	0.08	0.15	—	NA	1.3E+0	6.5E-5	0.015	0.002
ALPHA-CHLORDANE	3	32	0.09	0.05	0.08	—	NA	1.3E+0	6.0E-5	0.008	0.001
4,4'-DDT	9	32	0.28	0.12	0.25	—	NA	3.4E-1	—	0.007	—
ALPHA-BHC	6	33	0.18	0.09	0.18	—	NA	3.4E-1	—	0.005	—
BETA-BHC	2	32	0.06	0.05	0.01	—	NA	6.3E+0	—	0.005	—
4,4'-DDE	6	33	0.18	0.08	0.15	—	NA	3.4E-1	—	0.004	—
4,4'-DDD	2	32	0.06	0.07	0.13	—	NA	2.4E-1	—	0.003	—
TETRACHLOROETHENE	9	32	0.28	0.13	0.25	—	NA	5.2E-2	—	0.001	—

¹Shading indicates compounds selected for the Risk Assessment.

²Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

³Selected concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected). If the 95% UCL is greater than the maximum detected concentration, the selected concentration is the maximum detected concentration.

⁴95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

⁵Exceeds background concentrations. Background concentrations were considered in the ranking of inorganics and PAHs. A background concentration of zero was

TABLE A2-5
Warwick Area--Surface Soil

assumed during the ranking process for all other organic compounds. A 'Yes' indicates that the mean sample concentration exceeds the background concentration; these compounds are carried through the ranking process. A 'No' indicates the mean sample concentration does not exceed the background concentration; these compounds were eliminated as COPCs and were not carried through the ranking process.

⁶Cancer slope factor; NA - not applicable - no CSF is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

⁷Chronic reference dose; NA - not applicable - no RfD is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

⁸Relative ranking score--carcinogenic.

⁹Relative ranking score--noncarcinogenic.

TABLE A2-6
Warwick Area--Surface Soil

Ranking Process for Chemicals of Potential Concern Selection: Summary of Noncarcinogens
Ciba-Geigy Corporation, Cranston Rhode Island Site

Detected Compound ¹	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc. ² (mg/kg)	Selected Conc. ³ (mg/kg)	Bkg Conc. ⁴ (mg/kg)	>Bkg ⁵	CSF ⁶ (mg/kg-day) ⁻¹	RfD ⁷ (mg/kg-day)	RRS-C ⁸	RRS-N ⁹
ORGANICS											
PCB-1248	3	34	0.09	6.89	15.16	--	NA	--	3.0E-5	--	0.492
PCB-1254	15	32	0.47	2.86	5.19	--	NA	--	2.0E-5	--	0.253
2-NITROANILINE	2	31	0.06	7.15	7.00	--	NA	--	6.0E-5	--	0.114
METHOXYCHLOR	13	34	0.38	110.39	231.88	--	NA	--	5.0E-3	--	0.045
CHROMIUM	31	31	1.00	68.15	101.04	12.65	Yes	--	5.0E-3	--	0.020
HEPTACHLOR EPOXIDE	7	32	0.22	0.10	0.19	--	NA	9.1E+0	1.3E-5	0.137	0.014
ANTIMONY	6	23	0.26	2.27	5.35	0.00	Yes	--	4.0E-4	--	0.013
ZINC	28	31	0.90	2538.67	3911.97	75.94	Yes	--	3.0E-1	--	0.013
COPPER	30	31	0.97	118.02	225.23	14.99	Yes	--	3.7E-2	--	0.006
ISODRIN	2	32	0.06	0.08	0.16	--	NA	--	3.0E-5	--	0.005
CADMIUM	15	31	0.48	1.56	2.22	0.47	Yes	--	5.0E-4	--	0.004
NITROBENZENE	2	31	0.06	1.48	2.13	--	NA	--	5.0E-4	--	0.004
TINUVIN 327	8	25	0.32	6.34	9.27	--	NA	--	2.5E-3	--	0.004
DIELDRIN	5	32	0.16	0.08	0.16	--	NA	1.6E+1	5.0E-5	0.202	0.003
BARIUM	31	31	1.00	113.65	183.05	82.28	Yes	--	7.0E-2	--	0.003
GAMMA-CHLORDANE	9	32	0.28	0.08	0.15	--	NA	1.3E+0	6.5E-5	0.015	0.002
ALPHA-CHLORDANE	3	32	0.09	0.05	0.08	--	NA	1.3E+0	6.0E-5	0.008	0.001
ENDRIN ALDEHYDE	5	32	0.16	0.22	0.44	--	NA	--	3.0E-4	--	0.001
NICKEL	28	31	0.90	17.01	27.56	7.31	Yes	--	2.0E-2	--	0.001

¹Shading indicates compounds selected for the Risk Assessment.

²Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

TABLE A2-6
Warwick Area--Surface Soil

- ³Selected concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected). If the 95% UCL is greater than the maximum detected concentration, the selected concentration is the maximum detected concentration.
- ⁴95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.
- ⁵Exceeds background concentrations. Background concentrations were considered in the ranking of inorganics and PAHs. A background concentration of zero was assumed during the ranking process for all other organic compounds. A 'Yes' indicates that the mean sample concentration exceeds the background concentration; these compounds are carried through the ranking process. A 'No' indicates the mean sample concentration does not exceed the background concentration; these compounds were eliminated as COPCs and were not carried through the ranking process.
- ⁶Cancer slope factor; NA - not applicable - no CSF is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).
- ⁷Chronic reference dose; NA - not applicable - no RfD is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.
- ⁸Relative ranking score--carcinogenic.
- ⁹Relative ranking score--noncarcinogenic.

Attachment 3

Exposure Assessment Methodologies and Results

A3-1.0 Introduction

Exposure to the Chemicals of Potential Concern (COPC) was characterized using USEPA's *Risk Assessment Guidance for Superfund Human Health Evaluation Manual* (HHEM) methodology (USEPA, 1989). Many other resources were used in the exposure assessment and are cited where appropriate in the text and tables of this attachment. Two hypothetical exposure scenarios were evaluated: an on-site resident at the Warwick area and an on-site worker at the Production Area.

Soil is the only medium of interest in the on-site worker and on-site residential scenarios. Exposure is considered via the ingestion, dermal absorption, and inhalation routes. Air concentrations were modeled from the soil concentrations and consider COPC associated with fugitive dust and gaseous emissions. The soil-to-air transport model is included as Attachment 4 to the Risk Assessment.

A3-2.0 Methodologies

According to HHEM methodology, exposure is estimated as a daily intake (IN) in milligrams of chemical per kilogram of body weight (mg/kg-day) of the exposed receptor. The IN may result from exposure via ingestion, dermal absorption, and/or inhalation. Because exposure for the residential scenario includes both childhood and adult exposures, a time-weighted approach is taken using separate exposure input values for childhood and adult stages of life. The equation used for exposure via ingestion of a chemical associated with contaminated soil for the on-site worker and residential scenarios is as follows:

$$IN_{ing} = \frac{(CS \times IR_s \times CF \times FS \times EF \times ED)}{(BW \times AT)}$$

Where:

- IN_{ing} = Daily intake via ingestion
- CS = Concentration of chemical in soil (the lesser of the maximum detected concentration or the 95th percent upper confidence limit of the mean)
- IR_s = Soil ingestion rate

CF = Conversion factor
 FS = Fraction originating from contaminated source
 EF = Exposure frequency
 ED = Exposure duration
 BW = Body weight
 AT = Averaging time for exposure

The exposure equation for the soil ingestion pathway for the residential scenario includes both childhood and adult exposures. Thus, a time-weighted approach is taken, using separate values for childhood and adult exposure.

In both the on-site worker and on-site resident scenarios, exposure via dermal absorption was calculated using the following equation:

$$DAD = \frac{(CS \times CF \times SA_s \times FS \times AF \times ABS \times EF \times ED)}{(BW \times AT)}$$

Where:

DAD = Daily dermally absorbed dose
 SA_s = Skin surface area available for contact with soils
 AF = Soil-to-skin adherence factor
 ABS = Soil absorption fraction
 IN_{Der} = Daily intake via dermal absorption (adjusted for risk characterization)
 GAF = Gastrointestinal absorption fraction
 (Other variables are as previously described)

The DAD represents the dose absorbed by the body. As described in the risk characterization (see Attachment 6 of the Risk Assessment), reference doses (RfDs) and cancer slope factors (CSFs) are used to evaluate the risk associated with the calculated IN values. RfDs and CSFs are developed for the ingestion exposure route and are based on the ingested dose (IN_{Ing}). The gastrointestinally absorbed dose may be less than the IN_{Ing}. There are no RfD or CSF values based on the dermal absorption route. Because the DAD is an absorbed dose, the DAD must be adjusted so that it may be evaluated using an RfD or CSF. This is accomplished by dividing the DAD by the GAF. The resulting IN_{Der} is used during the risk characterization. Thus, the IN_{Der} may be viewed as an approximation of the ingestion intake necessary for the gastrointestinally

absorbed dose to equal the DAD. This method of adjusting the dermally absorbed dose is consistent with the approach described in the HHEM.

Inhalation exposure for the on-site worker and on-site residential scenarios were calculated using the following equation:

$$IN_{inh} = \frac{(CA \times FS \times EF \times ED \times IhR \times ET)}{(BW \times AT)}$$

Where:

- CA = Modeled concentration of chemical in the air. Model is described in Attachment 4 of the Risk Assessment. Modeled concentrations are based on the CS values described above.
- IN_{inh} = Daily intake via inhalation
- ET = Exposure time
- IhR = Inhalation rate
(Other variables are as previously described)

A3-3.0 Exposure Parameters

The USEPA (HHEM) recommends that a combination of upper-bound and average values be used in the exposure calculations. The exposure point concentrations used are upper-bound estimates (upper 95th percentile confidence limit on the mean) as described in Section A5.0 of the Risk Assessment. The other exposure parameters used to estimate chemical intakes are presented in Tables A3-3 through A3-14 of this attachment and summarized in Table A5-1 of the Risk Assessment text. These exposure parameters have been discussed previously with USEPA Region 1 and are described in the following paragraphs.

A3-3.1 Residential Scenario

Under the on-site residential scenario, exposure is assumed to occur over a 30-year period. It is assumed that 6 years are spent in early childhood (ages 1 through 6). The remaining 24 years of exposure are assumed to be spent as an adult. This 30-year exposure duration (ED) is a default value recommended by the USEPA (1991a) and represents an upper-bound estimate of the length

of time residents stay in one area. The distinction is made between early childhood and adult because the level of exposure experienced relative to body weight by a very young child is generally considered to be substantially greater than is experienced by an adult, particularly with regard to the ingestion of soil; such a difference is not believed to exist between an older child and an adult. The averaging time (AT) for exposure to noncarcinogenic chemicals is 10,950 days (8,760 days for adults and 2,190 days for children). This is equal to the ED. The AT for exposure to carcinogenic chemicals is 27,375 days. This is equal to the average lifetime of a receptor (75 years).

As recommended by the USEPA (1991a) it is assumed that a 70 kilogram (kg) adult ingests 100 milligrams (mg) of soil per day and a 15 kg child ingests 200 mg of soil per day. It is further assumed that 70% of this ingested soil comes from the contaminated area (FS=0.7). This fraction is based on the assumption that a residential receptor will average 8 hours away from the home each day (e.g., at work, shopping, visiting). The use of an FS value of 0.7 assumes that the person will consume soil at equal rates while at home and away from home. It is probable that a person consumes soil at a greater rate outside the home because 50% of the time that a person spends at home is while sleeping. Presumably, soil ingestion is minimal during sleep. Also, the FS value does not factor in soil associated with ingested food. For these reasons, an FS equal to 0.7 is probably conservative. This same discussion also applies to the dermal absorption route.

An adult inhalation rate (IhR) of 0.6 m³ per hour (USEPA, 1991b) is assumed. This is the average IhR for men and women engaged in light activity. Light activity includes most domestic work, personal care, hobbies, and conducting minor indoor repairs and home improvements. This value represents an average value for the part of the day spent at home, of which about 50% is spent sleeping. Inhalation rates will vary with activity; less during periods of rest (watching television, reading, sleeping), more during periods of higher activity (heavy cleaning, climbing stairs, exercising). An inhalation rate of 0.3 m³ per hour was assumed for children based on recommendations from the International Commission on Radiation Protection (1976).

An exposure time (ET) of 16 hours per day was assigned based on a USEPA (1990a) estimate that on average men and women spend 15.4 hours per day at home. It was assumed that young children will not be home alone, therefore, the ET for children is the same as that for adults.

Parameters specific to exposure via dermal absorption are the skin surface area available for contact (SA_s), soil-to-skin adherence factor (AF), soil absorption fraction (ABS), and

gastrointestinal absorption fraction (GAF). The USEPA (1992) recommended average SA_s for adults of 5000 cm^2 was used for this assessment. This value represents 25% of the total body surface area of adults (or approximately the hands, lower legs, forearms, neck, and head). This value assumes the receptor is wearing a short sleeved shirt and shorts (i.e., summer conditions) and does not allow for more clothing worn in the spring and fall by the Rhode Island resident. An SA_s for children of 2000 cm^2 was used for the Risk Assessment, as recommended by USEPA Region I.

A weighted approach was used regarding the soil-to-skin adherence factor (AF). This is because all of the studies we could find, including those referenced in the current USEPA dermal exposure assessment guidance (USEPA, 1992), are based on adherence to hands. As the guidance states, because hands generally have much greater contact with soil than do other parts of the body, AF values based on adherence to hands may overestimate the average adherence of soil to the entire exposed skin area. This is particularly true under this scenario where during the spring and fall most of the selected SA_s ($5,000\text{ cm}^2$) would be covered by clothing. Therefore, an AF of 0.5 was selected for the hands which in an average adult have a surface area of 800 cm^2 . An AF of 0.2 was selected for the remaining $4,200\text{ cm}^2$ of the body surface assumed to be in contact with soil.

The ABS and GAF for the dermal pathway are chemical specific. Absorption fractions and GAFs used are presented in Tables A3-9, and A3-10. These values represent upper-bound estimates of potential dermal absorption (ABS). The GAFs presented are generally the only values available for this parameter.

An exposure frequency (EF) of 350 days per year was used for inhalation exposures. This EF assumes that a resident will spend 15 days away from home on vacations, holidays, and weekend trips. This value does not take into account the potential reduction in air emissions resulting from snow cover and frozen ground in the winter. An EF of 230 days per year was used for exposure to soil (soil ingestion, dermal contact). This value assumes 15 days are spend away from home each year and that residents are not exposed to soil during 120 winter days per year when cold weather will cause a reduction in outdoor activities, an increase in the amount of clothing worn (thus decreasing dermal contact and hand to mouth soil transfer), and reducing the availability of soil due to snow cover and frozen ground. It is noted that the USEPA dermal

guidance suggests that a typical EF value for an adult who gardens one or two days per week during the warmer months is approximately 40 days/year (USEPA, 1992). If this is the case, then an EF of 230 days severely overestimates exposure.

A3-3.2 Worker Scenario

Under the on-site worker scenario, exposure is assumed to occur over a 25-year period. This 25-year ED is a default value recommended by the USEPA (1991a) and represents an upper-bound estimate of the length of time workers remain at one job. A standard 8-hour workday was assumed for the ET. The averaging time for exposure to noncarcinogenic chemicals is 9,125 days. This is equal to the exposure duration. The averaging time for exposure to carcinogenic chemicals is 27,375 days. This is equal to the average lifetime of a receptor (75 years).

As recommended by the USEPA (1991a) it is assumed that a 70 kg adult worker ingests 50 mg of soil per workday. The 50 mg of soil per workday is based on commercial and industrial workers who are routinely exposed to contaminated soil (USEPA, 1991). Based on the proposed use of the Site, we do not anticipate any future worker to be routinely exposed to contaminated soil. It is further assumed that 100% of this ingested soil comes from the contaminated area (FS=1.0). This fraction is based on the assumption that a worker will spend his/her entire workday in the contaminated area. This is likely to be an overestimate, since a worker probably will, on average, spend less than the entire workday at this area. The City of Cranston plans for this area to be used for vehicle parking, storage of snow removal equipment, and the storing and loading of road salt and sand. Although the Risk Assessment addresses the site in its current state, the Production Area will be covered with asphalt. This will virtually eliminate direct contact with the soil.

For inhalation exposures, an adult worker inhalation rate (IhR) of 1.4 m³ per hour is assumed (USEPA, 1990a). This is the average IhR for men and women engaged in moderate activity. Moderate activity includes such things as heavy cleaning and climbing stairs.

The USEPA (1992) recommended average SA_s for adults of 5000 cm² was used for this assessment. This value represents 25% of the total body surface area of adults (or approximately the hands, lower legs, forearms, neck, and head). This value assumes the receptor is wearing a short sleeved shirt and shorts (i.e., summer conditions) and does not allow for more clothing worn in the spring and fall. This weighted approach described above for the worker scenario was also applied to the residential scenario. An AF value of 0.5 mg/cm² was used for the hands, and

0.2 mg/cm² for the remainder of the body that is assumed to be in contact with the soil. The ABS and GAF are chemical-specific. Absorption fractions and GAFs used are presented in Table A3-4. These values represent upper-bound estimates of potential dermal absorption (ABS). The GAFs presented are generally the only values available for this parameter.

An EF of 80 days per year was used for all three routes of exposure. This represents five workdays per week for 17 winter weeks, minus five holidays, vacation days, and sick days during this period. These assumptions do not address the fact that exposure will be limited in the winter by snow cover, frozen ground, and heavy clothing.

A3-4.0 Results

As discussed in Attachment 2 of the Risk Assessment, COPC were selected separately for each Site area. The level of exposure associated with each COPC, measured in mg/kg-day, was estimated under each exposure scenario. Because the exposure assumptions differ somewhat, exposure levels of noncancer and cancer effects were calculated separately. Tables A3-1 and A3-2 summarize the exposure results of the Production Area and Warwick Area. The spreadsheet calculations tables, from which the values on Tables A3-1 and A3-2 were derived, are included as Tables A3-3 through A3-14.

Exposure results are combined with the appropriate criteria identified during the toxicity assessment (Section A6.0 of the Risk Assessment) to quantitatively characterize risks. The values shown in Tables A3-1 and A3-2 are carried into the risk characterization, included as Attachment 6. The values shown for the inhalation pathway include the combined contributions associated with fugitive dust and volatilization from soil to air.

**TABLE A3-1
EXPOSURE INTAKE SUMMARY
PRODUCTION AREA**

Noncancer Effects

Chemical	On-Site Worker		
	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)
PCB 1248	6.9×10^{-8}	1.0×10^{-7}	1.3×10^{-9}
PCB 1254	5.6×10^{-7}	8.4×10^{-7}	3.4×10^{-9}

Cancer Effects

Chemical	On-Site Worker		
	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)
PCB 1260	3.2×10^{-7}	4.7×10^{-7}	2.2×10^{-9}
gamma-Chlordane	6.8×10^{-9}	2.0×10^{-8}	2.3×10^{-10}

TABLE A3-2
EXPOSURE INTAKE SUMMARY
WARWICK AREA

Noncancer Effects

Chemical	Child Resident			Adult Resident		
	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation
PCB 1248	1.8×10^{-5}	2.8×10^{-6}	9.3×10^{-8}	7.6×10^{-6}	5.6×10^{-6}	1.6×10^{-7}
PCB 1254	6.1×10^{-6}	9.5×10^{-7}	8.8×10^{-9}	2.6×10^{-6}	2.0×10^{-6}	1.5×10^{-8}
2-Nitroaniline	8.2×10^{-6}	2.4×10^{-6}	9.2×10^{-8}	3.5×10^{-6}	4.9×10^{-6}	1.6×10^{-7}
Methoxychlor	2.7×10^{-4}	8.4×10^{-5}	5.1×10^{-8}	1.2×10^{-4}	1.7×10^{-4}	8.7×10^{-8}

Cancer Effects

Chemical	Child Resident			Adult Resident		
	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation
Aldrin	9.9×10^{-8}	3.0×10^{-8}	9.3×10^{-10}	4.2×10^{-8}	6.2×10^{-8}	1.6×10^{-9}
Beryllium	3.4×10^{-7}	8.8×10^{-7}	6.3×10^{-11}	1.5×10^{-7}	1.8×10^{-6}	1.1×10^{-10}
Dieldrin	7.5×10^{-8}	2.3×10^{-8}	8.3×10^{-10}	3.2×10^{-8}	4.7×10^{-8}	1.4×10^{-9}
Heptachlor epoxide	8.9×10^{-8}	2.7×10^{-8}	4.5×10^{-9}	3.8×10^{-8}	5.6×10^{-8}	7.6×10^{-9}
Total PCBs	8.5×10^{-6}	1.3×10^{-6}	4.1×10^{-8}	3.6×10^{-6}	2.7×10^{-6}	7.2×10^{-8}

TABLE A3-3
PRODUCTION AREA
ON-SITE WORKER SCENARIO
SOIL INGESTION PATHWAY

<i>Exposure Scenario</i>	CS (mg/kg)	IR _s (mg/day)	EF (days/yr)	FS	ED (yrs)	CF (kg/mg)	BW (kg)	AT (days)	INTAKE (mg/kg-day)
<i>Noncancer Effects</i>									
PCB 1248	0.44	50	80	1.0	25	1E-06	70	9,125	6.89E-08
PCB 1254	3.60	50	80	1.0	25	1E-06	70	9,125	5.64E-07
<i>Cancer Effects</i>									
PCB 1260	6.10	50	80	1.0	25	1E-06	70	27,375	3.18E-07
gamma-Chlordane	0.13	50	80	1.0	25	1E-06	70	27,375	6.78E-09
Total PCBs (a)	5.90	50	80	1.0	25	1E-06	70	27,375	3.08E-07

- a. A new data set was created based on the analytical results of PCB 1248, PCB 1254, and PCB 1260. Refer to Section A5.3.1 of the Risk Assessment.

TABLE A3-4
PRODUCTION AREA
ON-SITE WORKER SCENARIO
DERMAL ABSORPTION VIA SOIL PATHWAY

CHEMICAL	CS (mg/kg)	SA _s (cm ² /day)	AF (mg/cm ²)	ABS	FS	EF (days/yr)	ED (yrs)	CF (kg/mg)	BW (kg)	AT (days)	DAD (mg/kg-day)	GAF	IN _{Der} (mg/kg-day)
<i>Noncancer Effects</i>													
PCB 1248-hands	0.44	800	0.5	0.06(a)	1.0	80	25	1E-06	70	9,125	3.31E-08	1.0(a)	3.31E-08
PCB 1248-other	0.44	4,200	0.2	0.06(a)	1.0	80	25	1E-06	70	9,125	6.94E-08	1.0(a)	6.94E-08
PCB 1248-total	0.44	5,000	0.2/0.5	0.06(a)	1.0	80	25	1E-06	70	9,125	1.03E-07	1.0(a)	1.03E-07
PCB 1254-hands	3.60	800	0.5	0.06(a)	1.0	80	25	1E-06	70	9,125	2.71E-07	1.0(a)	2.71E-07
PCB 1254-other	3.60	4,200	0.2	0.06(a)	1.0	80	25	1E-06	70	9,125	5.68E-07	1.0(a)	5.68E-07
PCB 1254-total	3.60	5,000	0.2/0.5	0.06(a)	1.0	80	25	1E-06	70	9,125	8.39E-07	1.0(a)	8.39E-07
<i>Cancer Effects</i>													
PCB 1260-hands	6.10	800	0.5	0.06(a)	1.0	80	25	1E-06	70	27,375	1.53E-07	1.0(a)	1.53E-07
PCB 1260-other	6.10	4,200	0.2	0.06(a)	1.0	80	25	1E-06	70	27,375	3.21E-07	1.0(a)	3.21E-07
PCB 1260-total	6.10	5,000	0.2/0.5	0.06(a)	1.0	80	25	1E-06	70	27,375	4.74E-07	1.0(a)	4.74E-07
gamma-Chlordane-hands	0.13	800	0.5	0.10(b)	1.0	80	25	1E-06	70	27,375	5.43E-09	0.85(c)	6.39E-09
gamma-Chlordane-other	0.13	4,200	0.2	0.10(b)	1.0	80	25	1E-06	70	27,375	1.14E-08	0.85(c)	1.34E-08
gamma-Chlordane-total	0.13	5,000	0.2/0.5	0.10(b)	1.0	80	25	1E-06	70	27,375	1.68E-08	0.85(c)	1.98E-08
Total PCBs (d)-hands	5.90	800	0.5	0.06(a)	1.0	80	25	1E-06	70	27,375	1.48E-07	1.0(a)	1.48E-07
Total PCBs (d)-other	5.90	4,200	0.2	0.06(a)	1.0	80	25	1E-06	70	27,375	3.10E-07	1.0(a)	3.10E-07
Total PCBs (d)-total	5.90	5,000	0.2/0.5	0.06(a)	1.0	80	25	1E-06	70	27,375	4.58E-07	1.0(a)	4.58E-07

- a. This value was used as requested by USEPA Region I. Source: USEPA, 1995a.
- b. Source: No chemical-specific value could be found. Ryan et al. (1987) recommend a range of 0.01 to 0.10 for the dermal absorption of pesticides
- c. No chemical-specific value could be found. With the exception of benzo(a)pyrene, this is the lowest value listed for organic compounds (Jones and Owen, 1989).
- d. A new data set was created based on the analytical results of PCB 1248, PCB 1254, and PCB 1260. Refer to Section A5.3.1 of the Risk Assessment.

TABLE A3-5
 PRODUCTION AREA
 ON-SITE WORKER SCENARIO
 INHALATION PATHWAY - FUGITIVE DUST EMISSIONS

<i>Exposure Scenario</i>	CA ^a (mg/m ³)	IhR (m ³ /hr)	FS	ET (hr/event)	EF (days/yr)	ED (yrs)	BW (kg)	AT (days)	INTAKE (mg/kg-day)
<i>Noncancer Effects</i>									
PCB 1248	1.71E-09	1.4	1.0	8	80	25	70	9,125	6.00E-11
PCB 1254	1.40E-08	1.4	1.0	8	80	25	70	9,125	4.91E-10
<i>Cancer Effects</i>									
PCB 1260	2.38E-08	1.4	1.0	8	80	25	70	27,375	2.78E-10
gamma-Chlordane	5.07E-10	1.4	1.0	8	80	25	70	27,375	5.93E-12
Total PCBs (b)	2.30E-08	1.4	1.0	8	80	25	70	27,375	2.69E-10

- a. Modeled air concentrations resulting from the emission of dust-borne compounds in this area. The model is described in Attachment 4 of the Risk Assessment.
- b. A new data set was created based on the analytical results of PCB 1248, PCB 1254, and PCB 1260. Refer to Section A5.3.1 of the Risk Assessment.

TABLE A3-6
 PRODUCTION AREA
 ON-SITE WORKER SCENARIO
 INHALATION PATHWAY - VOLATILE EMISSIONS

CHEMICAL	CA ^a (mg/m ³)	IhR (m ³ /hr)	FS	ET (hr/event)	EF (days/yr)	ED (yrs)	BW (kg)	AT (days)	INTAKE (mg/kg-day)
<i>Noncancer Effects</i>									
PCB 1248	3.48E-08	1.4	1.0	8	80	25	70	9,125	1.22E-09
PCB 1254	8.24E-08	1.4	1.0	8	80	25	70	9,125	2.89E-09
<i>Cancer Effects</i>									
PCB 1260	1.61E-07	1.4	1.0	8	80	25	70	27,375	1.88E-09
<i>gamma</i> -Chlordane	1.96E-08	1.4	1.0	8	80	25	70	27,375	2.29E-10
Total PCBs (b)	2.78E-07(c)	1.4	1.0	8	80	25	70	27,375	3.25E-09

- a. Modeled air concentrations resulting from the volatile emissions of compounds in soil. Model is described in Attachment 4 of the Risk Assessment.
- b. Includes PCB 1248, PCB 1254, and PCB 1260.
- c. Although a new data set was created for Total PCBs where the concentrations of PCB 1248, PCB 1254, and PCB 1260 were summed separately for each sample (see Section A5.3.1 of the Risk Assessment), because the three PCBs have different volatilization rates, the CA value shown here is the sum of the CA values for PCB 1248, PCB 1254, and PCB 1260 shown above.

TABLE A3-7
WARWICK AREA
RESIDENTIAL SCENARIO
SOIL INGESTION PATHWAY
NONCANCER EFFECTS

Compound	CS (mg/kg)	IR _s (mg/day)	EF (days/yr)	FS	ED (yrs)	CF (kg/mg)	BW (kg)	AT (days)	INTAKE (mg/kg-day)
<i>Child</i>									
PCB 1248	15.0	200	230	0.7	6	1E-06	15	10,950	1.76E-05
PCB 1254	5.2	200	230	0.7	6	1E-06	15	10,950	6.12E-06
2-Nitroaniline	7.0	200	230	0.7	6	1E-06	15	10,950	8.23E-06
Methoxychlor	232.0	200	230	0.7	6	1E-06	15	10,950	2.73E-04
<i>Adult</i>									
PCB 1248	15.0	100	230	0.7	24	1E-06	70	10,950	7.56E-06
PCB 1254	5.2	100	230	0.7	24	1E-06	70	10,950	2.62E-06
2-Nitroaniline	7.0	100	230	0.7	24	1E-06	70	10,950	3.53E-06
Methoxychlor	232.0	100	230	0.7	24	1E-06	70	10,950	1.17E-04
<i>Combined Child and Adult</i>									
PCB 1248									2.52E-05
PCB 1254									8.74E-06
2-Nitroaniline									1.18E-05
Methoxychlor									3.90E-04

TABLE A3-8
WARWICK AREA
RESIDENTIAL SCENARIO
SOIL INGESTION PATHWAY
CANCER EFFECTS

Compound	CS (mg/kg)	IR _s (mg/day)	EF (days/yr)	FS	ED (yrs)	CF (kg/mg)	BW (kg)	AT (days)	INTAKE (mg/kg-day)
<i>Child</i>									
Aldrin	0.21	200	230	0.7	6	1E-06	15	27,375	9.88E-08
Beryllium	0.72	200	230	0.7	6	1E-06	15	27,375	3.39E-07
Dieldrin	0.16	200	230	0.7	6	1E-06	15	27,375	7.53E-08
Heptachlor epoxide	0.19	200	230	0.7	6	1E-06	15	27,375	8.94E-08
Total PCBs ^a	18	200	230	0.7	6	1E-06	15	27,375	8.47E-06
<i>Adult</i>									
Aldrin	0.21	100	230	0.7	24	1E-06	70	27,375	4.23E-08
Beryllium	0.72	100	230	0.7	24	1E-06	70	27,375	1.45E-07
Dieldrin	0.16	100	230	0.7	24	1E-06	70	27,375	3.23E-08
Heptachlor epoxide	0.19	100	230	0.7	24	1E-06	70	27,375	3.83E-08
Total PCBs ^a	18	100	230	0.7	24	1E-06	70	27,375	3.63E-06
<i>Combined Child and Adult</i>									
Aldrin									1.41E-07
Beryllium									4.84E-07
Dieldrin									1.08E-07
Heptachlor epoxide									1.28E-07
Total PCBs ^a									1.21E-05

a. A new data set was created based on the analytical results for PCB 1248 and PCB 1254, the only PCBs detected in Warwick Area soils. Refer to Section A5.3.1 of the Risk Assessment.

TABLE A3-9
WARWICK AREA
RESIDENTIAL SCENARIO
DERMAL ABSORPTION VIA SOIL PATHWAY
NONCANCER EFFECTS

Chemical	CS (mg/kg)	SA _s (cm ² /day)	AF (mg/cm ²)	ABS	FS	EF (days/yr)	ED (yrs)	CF (kg/mg)	BW (kg)	AT (days)	DAD (mg/kg-day)	GAF(a)	IN _{Der} (mg/kg-day)
<i>Child</i>													
PCB 1248-hands	15.0	400	0.5	0.06(b)	0.7	230	6	1E-06	15	10950	1.06E-06	1.00(b)	1.06E-06
PCB 1248-other	15.0	1600	0.2	0.06(b)	0.7	230	6	1E-06	15	10950	1.69E-06	1.00(b)	1.69E-06
PCB 1248-total	15.0	2,000	0.2/0.5	0.06(b)	0.7	230	6	1E-06	15	10,950	2.75E-06	1.00(b)	2.75E-06
PCB 1254-hands	5.2	400	0.5	0.06(b)	0.7	230	6	1E-06	15	10,950	3.67E-07	1.00(b)	3.67E-07
PCB 1254-other	5.2	1600	0.2	0.06(b)	0.7	230	6	1E-06	15	10,950	5.87E-07	1.00(b)	5.87E-07
PCB 1254-total	5.2	2,000	0.2/0.5	0.06(b)	0.7	230	6	1E-06	15	10,950	9.54E-07	1.00(b)	9.54E-07
2-Nitroaniline-hands	7.0	400	0.5	0.10(c)	0.7	230	6	1E-06	15	10,950	8.23E-07	0.90(d)	9.15E-07
2-Nitroaniline-other	7.0	1600	0.2	0.10(c)	0.7	230	6	1E-06	15	10,950	1.32E-06	0.90(d)	1.46E-06
2-Nitroaniline-total	7.0	2,000	0.2/0.5	0.10(c)	0.7	230	6	1E-06	15	10,950	2.14E-06	0.90(d)	2.38E-06
Methoxychlor-hands	232	400	0.5	0.10(c)	0.7	230	6	1E-06	15	10,950	2.73E-05	0.85(e)	3.21E-05
Methoxychlor-other	232	1600	0.2	0.10(c)	0.7	230	6	1E-06	15	10,950	4.37E-05	0.85(e)	5.14E-05
Methoxychlor-total	232	2,000	0.2/0.5	0.10(c)	0.7	230	6	1E-06	15	10,950	7.10E-05	0.85(e)	8.35E-05
<i>Adult</i>													
PCB 1248-hands	15.0	800	0.5	0.06(b)	0.7	230	24	1E-06	70	10,950	1.81E-06	1.00(b)	1.81E-06
PCB 1248-other	15.0	4200	0.2	0.06(b)	0.7	230	24	1E-06	70	10,950	3.81E-06	1.00(b)	3.81E-06
PCB 1248-total	15.0	5,000	0.2/0.5	0.06(b)	0.7	230	24	1E-06	70	10,950	5.63E-06	1.00(b)	5.63E-06
PCB 1254-hands	5.2	800	0.5	0.06(b)	0.7	230	24	1E-06	70	10,950	6.29E-07	1.00(b)	6.29E-07
PCB 1254-other	5.2	4200	0.2	0.06(b)	0.7	230	24	1E-06	70	10,950	1.32E-06	1.00(b)	1.32E-06
PCB 1254-total	5.2	5,000	0.2/0.5	0.06(b)	0.7	230	24	1E-06	70	10,950	1.95E-06	1.00(b)	1.95E-06
2-Nitroaniline-hands	7.0	800	0.5	0.10(c)	0.7	230	24	1E-06	70	10,950	1.41E-06	0.90(d)	1.57E-06
2-Nitroaniline-other	7.0	4200	0.2	0.10(c)	0.7	230	24	1E-06	70	10,950	2.96E-06	0.90(d)	3.29E-06
2-Nitroaniline-total	7.0	5,000	0.2/0.5	0.10(c)	0.7	230	24	1E-06	70	10,950	4.38E-06	0.90(d)	4.86E-06
Methoxychlor-hands	232.0	800	0.5	0.10(c)	0.7	230	24	1E-06	70	10,950	4.68E-05	0.85(e)	5.50E-05
Methoxychlor-other	232.0	4200	0.2	0.10(c)	0.7	230	24	1E-06	70	10,950	9.82E-05	0.85(e)	1.16E-04
Methoxychlor-total	232.0	5,000	0.2/0.5	0.10(c)	0.7	230	24	1E-06	70	10,950	1.45E-04	0.85(e)	1.71E-04

Chemical	CS (mg/kg)	SA _s (cm ² /day)	AF (mg/cm ²)	ABS	FS	EF (days/yr)	ED (yrs)	CF (kg/mg)	BW (kg)	AT (days)	DAD (mg/kg-day)	GAF(a)	IN _{Der} (mg/kg-day)
<i>Adult and Child Combined Combined - Total Exposure</i>													
PCB 1248													8.38E-06
PCB 1254													2.90E-06
2-Nitroaniline													7.24E-06
Methoxychlor													2.54E-04

- a. Source: Jones and Owen (1989), unless otherwise noted.
- b. Requested by USEPA Region 1. Source: USEPA, 1995a.
- c. Source: No chemical-specific values could be found. Ryan et al., (1987) recommend a range of 0.01 to 0.10 for the dermal absorption of semivolatile organics and pesticides bound in a soil matrix.
- d. No chemical-specific values could be found. Value shown is the lowest value listed in Jones and Owen (1989) for substituted benzene compounds.
- e. No chemical-specific values could be found. Value shown is the lowest value listed in Jones and Owen (1989) for organic compounds other than polycyclic aromatic hydrocarbons.

TABLE A3-10
WARWICK AREA
RESIDENTIAL SCENARIO
DERMAL ABSORPTION VIA SOIL PATHWAY
CANCER EFFECTS

Chemical	CS (mg/kg)	SA _s (cm ² /day)	AF (mg/cm ²)	ABS	FS	EF (days/yr)	ED (yrs)	CF (kg/mg)	BW (kg)	AT (days)	DAD (mg/kg-day)	GAF(a)	IN _{Der} (mg/kg-day)
<i>Child</i>													
Aldrin-hands	0.21	400	0.5	0.10(b)	0.7	230	6	1E-06	15	27,375	9.88E-09	0.85(c)	1.16E-08
Aldrin-other	0.21	1,600	0.2	0.10(b)	0.7	230	6	1E-06	15	27,375	1.58E-08	0.85(c)	1.86E-08
Aldrin-total	0.21	2,000	0.5/0.2	0.10(b)	0.7	230	6	1E-06	15	27,375	2.57E-08	0.85(c)	3.02E-08
Beryllium-hands	0.72	400	0.5	0.001(d)	0.7	230	6	1E-06	15	27,375	3.39E-10	0.001	3.39E-07
Beryllium-other	0.72	1,600	0.2	0.001(d)	0.7	230	6	1E-06	15	27,375	5.42E-10	0.001	5.42E-07
Beryllium-total	0.72	2,000	0.5/0.2	0.001(d)	0.7	230	6	1E-06	15	27,375	8.81E-10	0.001	8.81E-07
Dieldrin-hands	0.16	400	0.5	0.10(b)	0.7	230	6	1E-06	15	27,375	7.53E-09	0.85(c)	8.86E-09
Dieldrin-other	0.16	1,600	0.2	0.10(b)	0.7	230	6	1E-06	15	27,375	1.20E-08	0.85(c)	1.42E-08
Dieldrin-total	0.16	2,000	0.5/0.2	0.10(b)	0.7	230	6	1E-06	15	27,375	1.96E-08	0.85(c)	2.30E-08
Heptachlor epoxide-hands	0.19	400	0.5	0.10(b)	0.7	230	6	1E-06	15	27,375	8.94E-09	0.85(c)	1.05E-08
Heptachlor epoxide-other	0.19	1,600	0.2	0.10(b)	0.7	230	6	1E-06	15	27,375	1.43E-08	0.85(c)	1.68E-08
Heptachlor epoxide-total	0.19	2,000	0.5/0.2	0.10(b)	0.7	230	6	1E-06	15	27,375	2.32E-08	0.85(c)	2.73E-08
Total PCBs-hands (e)	18	400	0.5	0.06(f)	0.7	230	6	1E-06	15	27,375	5.08E-07	1.00(f)	5.08E-07
Total PCBs-other (e)	18	1,600	0.2	0.06(f)	0.7	230	6	1E-06	15	27,375	8.13E-07	1.00(f)	8.13E-07
Total PCBs-total (e)	18	2,000	0.5/0.2	0.06(f)	0.7	230	6	1E-06	15	27,375	1.32E-06	1.00(f)	1.32E-06
<i>Adult</i>													
Aldrin-hands	0.21	800	0.5	0.10(b)	0.7	230	24	1E-06	70	27,375	1.69E-08	0.85(c)	1.99E-08
Aldrin-other	0.21	4,200	0.2	0.10(b)	0.7	230	24	1E-06	70	27,375	3.56E-08	0.85(c)	4.18E-08
Aldrin-total	0.21	5,000	0.5/0.2	0.10(b)	0.7	230	24	1E-06	70	27,375	5.25E-08	0.85(c)	6.18E-08
Beryllium-hands	0.72	800	0.5	0.001(d)	0.7	230	24	1E-06	70	27,375	5.81E-10	0.001	5.81E-07
Beryllium-other	0.72	4,200	0.2	0.001(d)	0.7	230	24	1E-06	70	27,375	1.22E-09	0.001	1.22E-06
Beryllium-total	0.72	5,000	0.5/0.2	0.001(d)	0.7	230	24	1E-06	70	27,375	1.80E-09	0.001	1.80E-06

Chemical	CS (mg/kg)	SA _s (cm ² /day)	AF (mg/cm ²)	ABS	FS	EF (days/yr)	ED (yrs)	CF (kg/mg)	BW (kg)	AT (days)	DAD (mg/kg-day)	GAF(a)	IN _{Der} (mg/kg-day)
Dieldrin-hands	0.16	800	0.5	0.10(b)	0.7	230	24	1E-06	70	27,375	1.29E-08	0.85(c)	1.52E-08
Dieldrin-other	0.16	4,200	0.2	0.10(b)	0.7	230	24	1E-06	70	27,375	2.71E-08	0.85(c)	3.19E-08
Dieldrin-total	0.16	5,000	0.5/0.2	0.10(b)	0.7	230	24	1E-06	70	27,375	4.00E-08	0.85(c)	4.71E-08
Heptachlor epoxide-hands	0.19	800	0.5	0.10(b)	0.7	230	24	1E-06	70	27,375	1.53E-08	0.85(c)	1.80E-08
Heptachlor epoxide-other	0.19	4,200	0.2	0.10(b)	0.7	230	24	1E-06	70	27,375	3.22E-08	0.85(c)	3.79E-08
Heptachlor epoxide-total	0.19	5,000	0.5/0.2	0.10(b)	0.7	230	24	1E-06	70	27,375	4.75E-08	0.85(c)	5.59E-08
Total PCBs-hands (e)	18	800	0.5	0.06(f)	0.7	230	24	1E-06	70	27,375	8.71E-07	1.00(f)	8.71E-07
Total PCBs-other (e)	18	4,200	0.2	0.06(f)	0.7	230	24	1E-06	70	27,375	1.83E-06	1.00(f)	1.83E-06
Total PCBs-total (e)	18	5,000	0.5/0.2	0.06(f)	0.7	230	24	1E-06	70	27,375	2.70E-06	1.00(f)	2.70E-06
<i>Adult and Child Combined - Total Exposure</i>													
Aldrin													9.20E-08
Beryllium													2.68E-06
Dieldrin													7.01E-08
Heptachlor epoxide													8.32E-08
Total PCBs (e)													4.02E-06

- Source: Jones and Owen, 1989.
- Source: No chemical-specific values could be found. Ryan et al., (1987) recommend a range of 0.01 to 0.10 for the dermal absorption of pesticides and semivolatile organics bound in a soil matrix.
- No chemical-specific value could be found. Value shown is the lowest listed in Jones and Owen (1989) for organic compounds other than polycyclic aromatic hydrocarbons.
- Source: No chemical specific value could be found. Value shown is the average absorption of cadmium, the only inorganic for which a dermal absorption coefficient could be found (USEPA, 1992). If relative dermal absorption of cadmium and beryllium is similar to their relative oral absorption efficiencies (0.06 and 0.001, respectively - Jones and Owen, 1989), then the use of this dermal absorption value for beryllium is an overestimate and adds conservativeness to the exposure estimation.
- A new data set was created based on the analytical results for PCB 1248 and PCB 1254, the only PCBs detected in Warwick Area soils. Refer to Section A5.3.1 of the Risk Assessment.
- This value was requested by USEPA Region I. Source: USEPA, 1995a.

TABLE A3-11
WARWICK AREA
RESIDENTIAL SCENARIO
AIR INHALATION PATHWAY - FUGITIVE DUST EMISSIONS
NONCANCER EFFECTS

Chemical	CA (mg/m ³)	IhR (m ³ /hr)	ET (hrs/day)	EF (days/yr)	ED (yrs)	BW (kg)	AT (days)	INTAKE (mg/kg-day)
<i>Child</i>								
PCB 1248	5.34E-08	0.3	16	350	6	15	10,950	3.28E-09
PCB 1254	1.85E-08	0.3	16	350	6	15	10,950	1.14E-09
2-Nitroaniline	2.49E-08	0.3	16	350	6	15	10,950	1.53E-09
Methoxychlor	8.23E-07	0.3	16	350	6	15	10,950	5.05E-08
<i>Adult</i>								
PCB 1248	5.34E-08	0.6	16	350	24	70	10,950	5.62E-09
PCB 1254	1.85E-08	0.6	16	350	24	70	10,950	1.95E-09
2-Nitroaniline	2.49E-08	0.6	16	350	24	70	10,950	2.62E-09
Methoxychlor	8.23E-07	0.6	16	350	24	70	10,950	8.66E-08
<i>Combined</i>								
PCB 1248								8.90E-09
PCB 1254								3.08E-09
2-Nitroaniline								4.15E-09
Methoxychlor								1.37E-07

TABLE A3-12
WARWICK AREA
RESIDENTIAL SCENARIO
AIR INHALATION PATHWAY - FUGITIVE DUST EMISSIONS
CANCER EFFECTS

Chemical	CA (mg/m ³)	IhR (m ³ /hr)	ET (hrs/day)	EF (days/yr)	ED (yrs)	BW (kg)	AT (days)	INTAKE (mg/kg-day)
<i>Child</i>								
Aldrin	7.48E-10	0.3	16	350	6	15	27,375	1.84E-11
Beryllium	2.56E-09	0.3	16	350	6	15	27,375	6.28E-11
Dieldrin	5.70E-10	0.3	16	350	6	15	27,375	1.40E-11
Heptachlor epoxide	6.77E-10	0.3	16	350	6	15	27,375	1.66E-11
Total PCBs (b)	6.48E-08	0.3	16	350	6	15	27,375	1.59E-09
<i>Adult</i>								
Aldrin	7.48E-10	0.6	16	350	24	70	27,375	3.15E-11
Beryllium	2.56E-09	0.6	16	350	24	70	27,375	1.08E-10
Dieldrin	5.70E-10	0.6	16	350	24	70	27,375	2.40E-11
Heptachlor epoxide	6.77E-10	0.6	16	350	24	70	27,375	2.85E-11
Total PCBs (b)	6.48E-08	0.6	16	350	24	70	27,375	2.73E-09
<i>Combined</i>								
Aldrin								4.98E-11
Beryllium								1.71E-10
Dieldrin								3.80E-11
Heptachlor epoxide								4.51E-11
Total PCBs (b)								4.32E-09

- a. Modeled air concentrations resulting from the emission of dust-borne compounds in this area. The model is described in Attachment 4 of the Risk Assessment.
- b. A new data set was created based on the analytical results for PCB 1248 and PCB 1254, the only PCBs detected in Warwick Area soils. Refer to Section A5.3.1 of the Risk Assessment.

TABLE A3-13
WARWICK AREA
RESIDENTIAL SCENARIO
AIR INHALATION PATHWAY - VOLATILE EMISSIONS
NONCANCER EFFECTS

Chemical	CA ^a (mg/m ³)	IhR (m ³ /hr)	ET (hrs/day)	EF (days/yr)	ED (yrs)	BW (kg)	AT (days)	INTAKE (mg/kg-day)
<i>Child</i>								
PCB 1248	1.47E-06	0.3	16	350	6	15	10,950	9.02E-08
PCB 1254	1.24E-07	0.3	16	350	6	15	10,950	7.61E-09
2-Nitroaniline	1.48E-06	0.3	16	350	6	15	10,950	9.08E-08
Methoxychlor ^b	0.00E+00	0.3	16	350	6	15	10,950	0.00E+00
<i>Adult</i>								
PCB 1248	1.47E-06	0.6	16	350	24	70	10,950	1.55E-07
PCB 1254	1.24E-07	0.6	16	350	24	70	10,950	1.30E-08
2-Nitroaniline	1.48E-06	0.6	16	350	24	70	10,950	1.56E-07
Methoxychlor ^b	0.00E+00	0.6	16	350	24	70	10,950	0.00E+00
<i>Combined</i>								
PCB 1248								2.45E-07
PCB 1254								2.07E-08
2-Nitroaniline								2.47E-07
Methoxychlor ^b								0.00E+00

a. Modeled air concentrations resulting from the volatile emissions of compounds in soil.

Model is described in Attachment 4 of the Risk Assessment.

b. Methoxychlor is nonvolatile.

TABLE A3-14
WARWICK AREA
RESIDENTIAL SCENARIO
AIR INHALATION PATHWAY - VOLATILE EMISSIONS
CANCER EFFECTS

Chemical	CA ^a (mg/m ³)	IhR (m ³ /hr)	ET (hrs/day)	EF (days/yr)	ED (yrs)	BW (kg)	AT (days)	INTAKE (mg/kg-day)
<i>Child</i>								
Aldrin	3.73E-08	0.3	16	350	6	15	27,375	9.16E-10
Beryllium ^b	0.00E+00	0.3	16	350	6	15	27,375	0.00E+00
Dieldrin	3.32E-08	0.3	16	350	6	15	27,375	8.15E-10
Heptachlor epoxide	1.81E-07	0.3	16	350	6	15	27,375	4.44E-09
Total PCBs ^c	1.59E-06(d)	0.3	16	350	6	15	27,375	3.90E-08
<i>Adult</i>								
Aldrin	3.73E-08	0.6	16	350	24	70	27,375	1.57E-09
Beryllium ^b	0.00E+00	0.6	16	350	24	70	27,375	0.00E+00
Dieldrin	3.32E-08	0.6	16	350	24	70	27,375	1.40E-09
Heptachlor epoxide	1.81E-07	0.6	16	350	24	70	27,375	7.62E-09
Total PCBs ^c	1.59E-06(d)	0.6	16	350	24	70	27,375	6.69E-08
<i>Combined</i>								
Aldrin								2.49E-09
Beryllium								0.00E+00
Dieldrin								2.21E-09
Heptachlor epoxide								1.21E-08
Total PCBs ^c								1.06E-07

- a. Modeled air concentrations resulting from the volatile emissions of compounds in soil. Model is described in Attachment 4 of the Risk Assessment.
- b. Beryllium is nonvolatile.
- c. Includes PCB 1248 and PCB 1254.
- d. Although a new data set was created for Total PCBs where the concentrations of PCB 1248 and PCB 1254 are summed separately for each sample (see Section A5.3.1 of the Risk Assessment text); because these two PCBs have different volatilization rates, this CA value is the the sum of the CA values for PCB 1248 and PCB 1254 shown in Attachment 3, Table A3-13.

Attachment 4

Air Transport Analysis

A4-1.0 Introduction

An air transport analysis of contaminants of potential concern from the Ciba-Geigy Cranston, Rhode Island Site (the Site) was conducted to support a preliminary public health risk assessment. This attachment to the Risk Assessment presents a description of the methodology, data base, assumptions, and models used in the analysis.

The objective of the air transport analysis is to predict the maximum ground-level concentrations of the contaminants potentially released to the atmosphere from the Site. These predicted concentrations are representative of maximum long-term, on-site exposures associated with the potential land uses described in the risk assessment scenarios for hypothetical residents and hypothetical workers (See Section A5.0 of the Risk Assessment).

The analysis was conducted following guidelines established by the U.S. Environmental Protection Agency (USEPA) (USEPA, 1986a; 1987a; 1992a; 1993). A regulatory guideline air dispersion model was used to predict the maximum ground-level concentrations (USEPA, 1992b). Regulatory guidelines were also employed to predict air emission rates of each contaminant of potential concern (COPC) (USEPA, 1992a).

The air dispersion model accounts for the dilution and dispersion of contaminants from an emission source to a receptor considering site meteorological conditions. Site-specific data were used in the predictions of air emission rates and air concentrations whenever available. When site-specific data were not available, conservative assumptions were made so that health risks associated with the air pathway would not be underestimated.

A4-2.0 Site Description

The Site has been separated into three distinct areas for the purpose of investigating the magnitude and extent of possible chemical contamination. These areas are designated as:

- the Production Area,
- the Warwick Area, and
- the Waste Water Treatment Area.

The Production and Warwick Areas are addressed in the Risk Assessment and in this attachment. These areas are mostly covered by vegetation, concrete, and asphalt. A soil boring survey has been conducted for the areas and a number of different organic and inorganic chemical compounds have been detected in the soil samples. The aerial extent of assumed chemical contamination and the amount of soil cover (Houlday, 1994) for each designated area are provided in Table A4-1.

A4-3.0 Air Emissions Sources and Chemicals of Potential Concern

The Production, Warwick, and Waste Water Treatment Areas are a potential sources of gaseous emissions due to the evaporation of volatile and semivolatile compounds from the subsurface soil. Each area is also a potential source of wind blown dust contaminated by volatile, semivolatile, and nonvolatile compounds, including inhalable particulate matter (particle diameters $< 10 \mu\text{m}$; referred to as PM-10). The definition of volatile, semivolatile, and nonvolatile compounds and examples of the types of contaminants in each category are listed in Table A4- 2 (USEPA, 1990).

Separate COPC were selected for the Production and Warwick Areas (refer to Section A4.0 of the Risk Assessment). A list of COPC for each area along with their classification as volatile, semivolatile, or nonvolatile is provided in Table A4-3. The relevant physical and chemical properties of the COPC are provided in Table A4-4.

A4-4.0 Predicting Air Concentrations

A screening-type air dispersion modeling analysis was performed to predict long-term concentrations due to the area sources associated with the Site. The USEPA's Industrial Source Complex Short-Term (ISCST2) model (1992b) was used in the analysis. The ISCST2 model is a regulatory guideline air dispersion model and is designated as the preferred model for predicting concentrations from complicated sources such as area sources (USEPA, 1986a; 1987a; 1993). The model is based on the Gaussian plume equations to predict concentrations from continuous

sources. For this analysis, it was assumed that emissions of COPC do not undergo any chemical reactions in the atmosphere and that no removal processes, such as wet or dry deposition, act on the plume during its transport from the source.

The ISCST2 model can perform multiple source short-term concentration predictions on square area sources. Data required to run the model include source characteristics, meteorology, and receptor grid locations.

The ISCST2 model was run to predict concentrations representative of maximum long-term on-site exposures associated with the potential land uses described in the risk assessment scenarios for hypothetical residents and hypothetical workers. This was accomplished by assuming each area of the Site is configured as a square with a receptor located in the center of the square. The ISCST2 model is not capable of predicting concentrations within an area source. Therefore, each designated area of the Site was subdivided so that the receptor was located at the edge of four square emission sources as illustrated in Figure A4-1 for the Production Area. Similar source configurations were used in the ISCST2 model to predict concentrations representative of receptors located in the center of the Warwick Area.

The characteristics of the area sources required as input to the ISCST2 model include emission rates, location coordinates, emission release height above ground, and the length of a side of a square area. Concentration predictions are directly proportional to the emission rate entered in the ISCST2 model. To simplify the air dispersion modeling analysis, a unit emission rate of $1 \mu\text{g/s-m}^2$ was used in the ISCST2 model for each of the four sources representing the Production Area and the Warwick Area. By inputting a unit emission rate of $1 \mu\text{g/s-m}^2$, the results obtained from the ISCST2 model are unit concentrations (i.e., $\mu\text{g/m}^3$ per $\mu\text{g/s-m}^2$).

Compound-specific concentrations can then be determined based on the unit concentrations times the compound-specific area source emission rates. The prediction of area source emission rates is described in the next section. A summary of the source characteristics used for modeling each designated area of the Site is presented in Table A4.5.

Meteorology required as input to the ISCST2 model include wind speed, wind direction, and Pasquill atmospheric stability category. Ambient air temperature and mixing height values are also required, but these parameters have an insignificant effect on concentration predictions for the Cranston Site. Meteorology representative of annual average conditions were used in the air

dispersion modeling so that the concentration predictions are representative of annual averages. Annual meteorology is characterized by neutral (D) stability and a mean wind speed of 4.74 m/s (Bair, 1992). The mean annual wind speed is based on measurements made by the National Weather Service in Providence. A worst-case wind direction was determined for each source configuration by varying the wind direction in 10° increments and selecting the highest concentration prediction.

The receptor grid locations input to the ISCST2 model were at ground-level in the center of each designated area of the Cranston site.

The unit concentrations predicted by the ISCST2 model are presented in Table A4-6.

A4-5.0 Predicting Air Emission Rates

A4-5.1 Introduction

The current methodologies recommended by the USEPA for predicting emissions to the atmosphere from a contaminated site are contained in *Guideline for Predictive Baseline Emissions Estimation Procedures for Superfund Sites*, (USEPA 1992a). This document contains procedures for estimating:

- A) Gaseous emissions from subsurface soils;
- B) Gaseous emissions from nonaerated surface impoundments and contaminants in solution pooled at soil surfaces;
- C) Volatile nonmethane organic compound emissions from codisposal landfills (i.e., toxic wastes in combination with municipal or sanitary wastes);
- D) Free-phase volatile contaminants directly exposed to the atmosphere; and
- E) Solid and semivolatile compounds emitted as particulate matter.

Only items A and E are applicable to the Site. Gaseous emissions may be released due to the evaporation of volatile and semivolatile contaminants in the subsurface soil (Item A). In addition, volatile, semivolatile, and nonvolatile contaminants may be released as constituents of

particulate matter emissions due to wind erosion of exposed soil surfaces (Item E). The emission rate models recommended by the USEPA (1992a) predict air emission rates as a function of contaminant concentration and contaminant physical and chemical properties within the soil. The modeling methodology, data, and assumptions used to predict contaminant air emission rates are described in the following.

A4-5.2 Air Emissions from Subsurface Soils

Preferably, soil gas measurements are used to predict the air release potential of volatile and semivolatile contaminants from subsurface soils. In the absence of soil gas measurements, soil bulk concentrations can be used for predicting the air release potential of contaminants. For the Site, soil gas measurements have not been made, whereas soil bulk concentrations have been determined from an on-site soil boring survey.

The first step in determining air emission rates based on soil bulk concentrations (C_{soil}) is to determine if free-phase volatile and semivolatile contaminants exist in the soil vadose zone as a liquid-phase waste layer or discrete film. The vadose zone is that region above the water table or saturated zone of the subsurface soil. Free-phase contaminants in the vadose zone are indicated if C_{soil} is greater than the saturation concentration (C_{sat}). Under the alternative scenario, where C_{soil} is less than C_{sat} , all contaminants are assumed to be fully incorporated in the vadose zone soil matrix (i.e., in solution with the available soil moisture and adsorbed to the soil particles). It is further assumed for this scenario that no discrete waste layers or films were evident in the soil samples. An illustration of the two scenarios is given in Figure A4-2.

Separate procedures are required to calculate air emission rates for free-phase contaminants ($C_{\text{soil}} > C_{\text{sat}}$) and fully incorporated contaminants ($C_{\text{soil}} < C_{\text{sat}}$) in the soil vadose zone.

A4-5.2.1 Saturation Concentration Calculations

The USEPA (1992a) provides an equation for calculating C_{sat} as a function of the soil/water partition coefficient (K_d in l/kg or ml/g); the solubility of the contaminant in water (s in mg/l-water); and the soil moisture content (θ_m in l-water/kg-soil or ml-water/g-soil):

$$C_{\text{sat}} = (K_d \times s \times n_m) + (s \times \theta_m) \quad (\text{A4-1})$$

where n_m is the soil moisture content expressed as a weight fraction (kg-water/kg-soil). The

parameter values used as input to Equation (A4-1) for the Site are presented in the following discussion.

Values of K_d were estimated based on the following equation (USEPA, 1992a):

$$K_d = K_{oc} \times f_{oc} \quad (A4-2)$$

where K_{oc} is the soil/water partition coefficient (l/kg or ml/g) and f_{oc} is the fraction of organic content in the soil (mg/mg). The default value of f_{oc} is 0.02 (USEPA, 1992a). Values of K_{oc} for each COPC are provided in Table A4-4.

As indicated in Table A4-4, K_{oc} values for 2-nitroaniline and aniline were not found in the literature. For these COPCs, K_{oc} values were calculated based on the octanol/water partition coefficient, K_{ow} (l/kg or ml/g), using the following equation recommended by the USEPA (1992a):

$$K_{oc} = 10^{0.544 \log K_{ow} + 1.377} \quad (A4-3)$$

This equation is based on a wide variety of contaminants, mostly pesticides. Table A4-4 provides the log K_{ow} values used in the equation.

Also provided in Table A4-4 is the solubility of each COPC in water. Site-specific data on the moisture content of the soils were not readily available. A typical value of 20% moisture content for loam (Wanielista, 1990) was used in the analysis. Equivalent values of n_m and θ_m are 0.2 kg/kg and 0.2 l/kg, respectively.

Table A4-7 presents a summary of the worksheet for calculating C_{sat} values for each volatile and semivolatile COPC in each designated area. Also provided in the table are C_{soil} values obtained from the on-site soil boring survey. The table allows for the ready comparison of C_{soil} and C_{sat} values. For all volatile and semivolatile COPCs at the Site, the soil bulk concentrations are less than the saturation concentrations. This indicates that the COPC are fully incorporated in the

vadose zone soil matrix. The procedures required to calculate air emission rates for this scenario are described in the following.

A4-5.2.2 Air Emission Rate Calculations for $C_{soil} < C_{sat}$

The results of the soil boring survey indicate that the volatile and semivolatile COPC at the Site are fully incorporated in the vadose zone soil matrix. The USEPA (1992a) provides an equation for predicting air emission rates from contaminated subsurface soil when the measured soil bulk concentrations are less than the saturated concentration. The average air emission rate (E in g/s) of a component for a specific exposure time [t in second(s)] is a function of the exposed surface area (A in cm^2); the effective diffusivity of the component in air (D_e in cm^2/s); the soil porosity (ϵ); the soil/air partition coefficient (K_{as} in g/cm^3); and the soil bulk concentration (C_{soil}) of the component:

$$E = \frac{2AD_e\epsilon K_{as}C_{soil}}{\sqrt{\pi\alpha t}} \quad (A4-4)$$

An estimate of the exposed surface areas of the Site is provided in Table A4-1. The effective diffusivity of the component is calculated based on the component's diffusion coefficient in air (D in cm^2/s) and the soil porosity (USEPA, 1992a):

$$D_e = D\epsilon^{0.33} \quad (A4-5)$$

Diffusion coefficients in air for each COPC are provided in Table A4-4. The air diffusion coefficients of *gamma*-chlordane, 2,3,7,8-TCDF, and 2,4-dichlorophenol were calculated using the USEPA's CHEM7 chemical compound property processor (1991). When the soil is wet more often than dry, it is appropriate to use the air-filled soil porosity (P_a) in Equations (A4-4) and (A4-5) to determine emission rates and effective diffusivities (USEPA, 1992a). The air-filled soil porosity is calculated by:

$$P_a = P_t - \theta_m \beta \quad (A4-6)$$

where P_t is the total soil porosity (dimensionless), θ_m is the soil moisture content (ml/g), and β is the soil bulk density (g/cm^3). The total soil porosity for the Cranston site is 0.42 based on soil boring measurements. A typical soil moisture content for loam is 0.2 ml/g (Wanielista, 1990).

The default value for β is 1.5 g/cm³ (USEPA, 1992a). Using these values in Equation (A4-6) yields an air-filled soil porosity of 0.12.

The soil/air partition coefficient of a component is calculated based on the component's soil/water partition coefficient [refer to Equation (A4-2)] and Henry's Law constant (H in atm-m³/mole) (USEPA, 1992a):

$$K_{as} = \frac{H}{K_d} \times 41 \quad (\text{A4-7})$$

where 41 is a conversion factor to change H to dimensionless form. Values of K_d and H for each COPC are provided in Table A4-4.

Soil bulk concentrations are provided in Table A4-7. The parameter α is a function of the effective diffusivity, soil porosity, particle density (ρ in g/cm³), and the soil/air partition coefficient (USEPA, 1992a):

$$\alpha = \frac{D_e \epsilon}{\epsilon + \rho(1 - \epsilon)/K_{as}} \quad (\text{A4-8})$$

The default value for particle density is 2.65 g/cm³ (USEPA, 1992a). Values of D_e and K_{as} were calculated using Equations (A4-5) and (A4-7), respectively. The air-filled soil porosity is 0.12.

Exposure time (t in Equation A4-4) is assumed to be 30 years (USEPA, 1992a) which is equivalent to 9×10^8 seconds.

Table A4-8 presents a summary of the worksheet for predicting air emission rates of volatile and semivolatile COPC from assumed contaminated subsurface soil where $C_{\text{soil}} < C_{\text{sat}}$. Air emission rates are provided in units of g/s and g/s-m². The latter emission rates are area source emission rates which are needed for input to the air dispersion model.

A4-5.3 Air Emissions from Wind Erosion of Exposed Soil Surfaces

Although the Site is substantially covered by vegetation, concrete, and asphalt, it does not contain 100% unbroken soil cover. An estimate of the fraction of soil cover of the Site is given in Table A4-1 (Woodward-Clyde Consultants, 1994). As the worst-case, it is assumed that the exposed soil surfaces do not contain any hardened crust. Therefore, there is a potential for wind erosion of exposed soil surfaces.

Currently there are two methodologies recommended by the USEPA (1992a) for predicting volatile, semivolatile, and nonvolatile contaminant emissions as constituents of particulate matter due to wind erosion of exposed soil surfaces: 1) the unlimited reservoir model, and 2) the limited reservoir model. The appropriate model is selected based on the threshold friction velocity (u_t). The threshold friction velocity is the minimum wind speed needed to suspend erodible soil particles. The lower the threshold friction velocity, the higher the potential for soil erosion by the wind. If the threshold friction velocity (corrected for nonerodible elements) is less than or equal to 0.75 cm/s, then the soil is classified as having unlimited erosion potential and the unlimited reservoir model should be used. If the threshold friction velocity (corrected for nonerodible elements) is greater than 0.75 cm/s, then the soil is classified as having limited erosion potential and the limited reservoir model should be used.

A4-5.3.1 Determining the Threshold Friction Velocity

The threshold friction velocity is determined from an empirical relationship of the mode of the surface soil aggregate size distribution. The aggregate size distribution mode is the particle size containing the highest percentage of material from a representative surface soil sample. Size distribution data of surface soil samples for the Site are available from the soil boring survey. The data are summarized as particle sizes (mm) for which 10%, 50%, 60%, and 90% of the soil sample is finer. These data are plotted in Figures A4-3 and A4-4 for the Production Area and the Warwick Area, respectively.

The data were analyzed to determine the mode of the distribution for size ranges recommended by the USEPA (1992a): > 4 mm; 2 to 4 mm; 1 to 2 mm; 0.5 to 1 mm; 0.25 to 0.5 mm; and < 0.25 mm. The data indicate that most of the surface soil samples are made up of particles with sizes less than 0.25 mm. The mode in the aggregate distribution lies between 0 and 0.25 mm. The aggregate size distribution mode is taken to be 0.125 mm.

The threshold friction velocity is determined from the empirical relationship with the aggregate

size distribution mode as given in Figure A4-5. The appropriate value of u_t is 27.5 cm/s for an aggregate size distribution mode of 0.125 mm. A factor (C_f) is used to correct for the nonerodible elements (e.g., stones, clumps of grass, etc.) in the surface soil. Where site-specific data are not available to determine an appropriate value of C_f , a conservative default value of 1.5 is recommended by the USEPA (1992a). The corrected threshold friction velocity (u_t^*) is

$$\begin{aligned} u_t^* &= u_t \times C_f \\ u_t^* &= 27.5 \text{ cm / s} \times 1.5 \\ u_t^* &= 41.25 \text{ cm / s} \end{aligned} \quad (\text{A4-9})$$

Since u_t^* is less than 75 cm/s, the unlimited reservoir model was selected to predict contaminant emission rates as constituents of particulate matter due to wind erosion of exposed soil surfaces.

A4-5.3.2 The Unlimited Reservoir Model

The annual average emission rate (E_{10} in g/s-m²) for each contaminant emitted as inhalable particulate matter from wind erodible surface soil is predicted using the following equation (USEPA, 1992a):

$$E_{10} = 0.00001(1-V) \left(\frac{\bar{u}}{u_t} \right)^3 F(x) C_{surf}^* \quad (\text{A4-10})$$

where V is the fraction of assumed contaminated surface with continuous vegetative cover; \bar{u} is the mean annual wind speed at 10 m anemometer height (m/s); u_t is the equivalent threshold value of wind speed at 7 m anemometer height (m/s); $F(x)$ is an empirical function of the unlimited reservoir model; and C_{surf} is the fractional percent by weight of the component from bulk samples of surface soil.

An estimate of the fraction of assumed contaminated surface with continuous soil cover for each of the designated areas of the Site is given in Table A4-1. The mean annual wind speed at 10 m anemometer height is 4.74 m/s based on measurements made by the National Weather Service in Providence, Rhode Island (Bair, 1992). The equivalent threshold value of wind speed at 7 m anemometer height is calculated based on the following equation provided by the USEPA (1992a):

$$\begin{aligned}
 u_t &= 18.1 \frac{u_t}{100} \\
 u_t &= 18.1 \frac{0.4125 \text{ m/s}}{100} \\
 u_t &= 5.10 \text{ m/s}
 \end{aligned}$$

(A4-11)

The value of function F(x) is 1.65 based on the curve presented in Figure A4-7 (USEPA, 1992a) where:

$$\begin{aligned}
 x &= 0.886 \frac{u_t}{u} \\
 x &= 0.886 \frac{5.10 \text{ m/s}}{4.74 \text{ m/s}} \\
 x &= 0.953
 \end{aligned}$$

(A4-12)

The concentrations of each volatile, semivolatile, and nonvolatile COPC in the surface soils for each designated area of the Site are presented in Table A4-9. These values were obtained from the on-site soil boring survey. Table A4-9 also presents a summary of the worksheet for predicting air emission rates of each COPC emitted as inhalable particulate matter from wind erodible surface soil.

A4-6.0 Uncertainties in the Air Transport Analysis

Atmospheric dispersion models are reasonably reliable in predicting the magnitude of the highest concentrations occurring at some time at some location within a given area of interest. The USEPA (1986a) reports errors in the highest predicted concentrations of 10 to 40 percent to be typical. To offset the inherent uncertainties in the air transport analysis, a number of conservative assumptions were made that led to overestimation of the maximum concentrations.

Annual meteorology was characterized as a single event of neutral atmospheric stability, a mean wind speed of 4.74 m/s, and a constant worst-case wind direction for each designated area.

These assumptions will tend to overestimate actual maximum long-term concentrations because they do not account for the highly variable meteorological conditions that will occur at the Site over a long period of time.

Several assumptions were made in predicting air emission rates that will tend to overestimate actual maximum long-term concentrations. First, the aeral extent of assumed chemical contamination was overstated. In addition, the concentrations of COPC were overstated by assuming the 95th percent upper confidence limit of the mean concentrations in site soils are distributed throughout the assumed contaminated area. It was also assumed that the exposed soil surfaces do not contain any hardened crust. This assumption tends to overestimate the amount of contaminants released from the site as particulate matter due to wind erosion of exposed soil surfaces.

A4-7.0 Results of the Air Transport Analysis

The results of the air transport analysis are summarized in Table A4-10. This table provides the predicted ambient air concentrations of each COPC for each of the designated areas of the Site. The predicted ambient air concentrations are representative of the maximum long-term on-site exposures associated with the potential land uses described in the risk assessment scenarios for hypothetical residents and hypothetical workers.

The results presented in Table A4-10 are based on the unit concentrations obtained from the USEPA's ISCST2 model (refer to Table A4-6) multiplied by the appropriate area source emission rate (refer to Tables A4-8 and A4-9). Each designated area of the Site is a potential source of gaseous emissions due to the evaporation of volatile and semivolatile compounds from the subsurface soil and wind blown dust contaminated by volatile, semivolatile, and nonvolatile compounds. The ambient concentration consists of the contributions due to these two emission release mechanisms. Table A4-10 summarizes the individual concentration components as well as the combined ambient concentrations.

A4-8.0 References

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Electronic Handbook Publishers, Inc., 1994, *Electronic Handbook of Risk Assessment Values*, Bellvue, Washington.

USEPA, 1986a, *Guideline on Air Quality Models*, Revised, EPA-450/2-78-027R, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina 27711.

USEPA, 1986b, *Superfund Public Health Evaluation Manual*, EPA-540/1-86-060, Office of Emergency Remedial Response, Washington, DC.

USEPA, 1987a, *Supplement A to the Guideline on Air Quality Models*, Revised, EPA-450/2-78-027R, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina 27711.

USEPA, 1987b, *Hazardous Waste Treatment, Storage, and Disposal Facilities (TSDF) - Air Emission Models*, EPA-450/3-97-026, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina.

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Wanielista, Martin, 1990, *Hydrology and Water Quality Control*, John Wiley & Sons, Inc., New York, New York.

Woodward-Clyde Consultants, 1994, personal communication from Mark Houlday (WCC) to Tom Marshall (PTRL), based on on-site observations by WWC personnel.

TABLE A4-I Areas of Potential Contamination.

Area Designation	Area		Fraction of Soil Cover ^a
	(ft ²)	(m ²)	
Production	140,000	13,000	0.90
Warwick	35,000	3,250	0.90

^a(Woodward-Clyde Consultants, 1994).

TABLE A4-2. Volatile, Semivolatile, and Nonvolatile Compounds.

Volatile Compounds (> 1 mmHg vapor pressure at 25 °C)

- All monochlorinated solvents. Also trichloroethylene, trichloroethane, tetrachloroethane.
- Most simple aromatic solvents: e.g., benzene, xylene, toluene, and ethylbenzene.
- Most alkanes up to decane (C₁₀)
- Inorganic gases: e.g., hydrogen sulfide, chlorine, and sulfur dioxide.

Semivolatile Compounds (10⁻⁷ to 1 mmHg vapor pressure at 25 °C)

- Most polychlorinated biphenyls, dichlorobenzenes, aniline, nitroaniline, and phthalates.
- Most pesticides: e.g., dieldrin, toxaphene, and parathion.
- Most complex alkanes: dodecane and octadecane.
- Most polynuclear aromatics: e.g., naphthalene, phenanthrene, and benz(a)anthracene.
- Mercury.

Nonvolatile Compounds or Particulate Matter (<10⁻⁷ mmHg vapor pressure at 25 °C)

- Larger polynuclear aromatics: e.g., chrysene.
 - Metals: e.g., lead and chromium.
 - Other inorganic compounds: e.g., asbestos, arsenic, and cyanides.
-

TABLE A4-3. List of Chemicals of Potential Concern (COPC).

Area	Chemical	CAS Number	Vapor Pressure (mmHg)	Classification
Production	PCB 1248	12672-29-6	1.80E-04	Semivolatile
	PCB 1254	11097-69-1	4.30E-05	Semivolatile
	PCB 1260	11096-82-5	1.10E-05	Semivolatile
	<i>gamma</i> -Chlordane	57-74-9	2.50E-05	Semivolatile
Warwick	PCB 1248	12672-29-6	1.80E-04	Semivolatile
	PCB 1254	11097-69-1	4.30E-05	Semivolatile
	2-Nitroaniline	88-74-4	3.00E-03	Semivolatile
	Methoxychlor	72-43-5	4.96E-09	Nonvolatile
	Aldrin	309-00-2	1.24E-04	Semivolatile
	Beryllium	7440-41-7	0	Nonvolatile
	Dieldrin	60-57-1	1.78E-07	Semivolatile
	Heptachlor Epoxide	1024-57-3	3.00E-04	Semivolatile

TABLE A4-4. Chemical Properties.

Chemical		CAS Number	MW Molecular Weight (g/g-mole)	p Vapor Pressure (mmHg)	D Diffusion Coefficient in Air (cm ² /s)	H Henry's Law Constant (atm-m ³ /mole)	s Solubility in Water (mg/l-water)	K _{oc} Organic Carbon Partition Coeff. (ml/g)	log K _{ow} Log of the Octanol/Water Partition Coeff. (mg/l)
PCB 1248	(1)	12672-29-6	288	0.00018	0.05498	0.0004	0.2	277000	6.11
PCB 1254	(1)	11097-69-1	328	0	0.05251	0.0002	0.041	2140000	6.94
PCB 1260	(1)	11096-82-5	372	0	0.04909	0.00025	0.0144	6700000	6.91
gamma-Chlordane	(1)	57-74-9	409.8	0	0.045	0.000048	0.006	9500	4.78
2-Nitroaniline	(2)	88-74-4	138.14	0.003	0.073	0	1.26E+03 (3)	236 (4)	1.83 (3)
Methoxychlor	(1)	72-43-5	345.65	0	0.04121	0	0.1	80000	4.3
Aldrin	(1)	309-00-2	365	0.00012	0.04744	0.000496	0.18	96000	3.01
Beryllium	(1)	7440-41-7	9.01	0	0	0	0		
Dieldrin	(1)	60-57-1	380.95	0	0.04875	0.000011	0.195	1700	3.5
Heptachlor Epoxide	(1)	1024-57-3	389	0.0003	0.04596	0.000032	0.35	220	2.7

References:

(1)Electronic Handbook Publishers, Inc., 1994, "Electronic Handbook of Risk Assessment Values", Bellevue, Washington.

(2)U.S. EPA, 1987b, "Hazardous Waste Treatment, Storage, and Disposal Facilities (TSDF) - Air Emission Models", EPA-450/3-97-026, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina.

(3)U.S. EPA, 1994, "Risk Reduction Engineering Laboratory (RREL) Treatability Database", contained on the Alternative Treatment Technology Information Center (ATTIC) Bulletin Board System.

(4)Calculated value from Log K_{ow}.

TABLE A4-5. Source Characteristics Used in the ISCST2 Model.

Designated Area (Source No.)	Emission Rate ($\mu\text{g/s-m}^2$)	Location Coordinates		Emission Release Height Above Ground (m)	Length of a Side of a Square Area (m)
		X (m)	Y (m)		
Production Area					
Source No. 1	1	-56.9	0	0	56.9
Source No. 2	1	0	0	0	56.9
Source No. 3	1	0	-56.9	0	56.9
Source No. 4	1	-56.9	-56.9	0	56.9
Warwick Area					
Source No. 1	1	-28.5	0	0	28.5
Source No. 2	1	0	0	0	28.5
Source No. 3	1	0	-28.5	0	28.5
Source No. 4	1	-28.5	-28.5	0	28.5

Table A4-6. ISCST2 Model Results.

Designated Area of the Cranston Site	Unit Concentrations ($\mu\text{g}/\text{m}^3$ per $\mu\text{g}/\text{s}\cdot\text{m}^2$)
Production Area	2.94176
Warwick Area	2.6884

TABLE A4-7. C_{sat} Calculation Worksheet for Volatile and Semivolatile Contaminants.

Contaminant	CAS Number	K_d (ml/g)	K_{oc} (ml/g)	$\log K_{ow}$ (ml/g)	f_{oc} (mg/mg)	s (mg/l-water)	n_m (kg/kg)	q_m (l/kg)	C_{sat} (mg/kg)	C_{soil} (mg/kg)
Production Area										
PCB 1248	12672-29-6	5540	277000	6.11	0.02	2.00E-01	0.2	0.2	222	0.21
PCB 1254	11097-69-1	42800	2140000	6.94	0.02	4.10E-02	0.2	0.2	351	2.00
PCB 1260	11096-82-5	134000	6700000	6.91	0.02	1.44E-02	0.2	0.2	386	6.40
<i>gamma</i> -Chlordane	57-74-9	190	9500	4.78	0.02	6.00E-03	0.2	0.2	0.229	0.070
Warwick Area										
PCB 1248	12672-29-6	5540	277000	6.11	0.02	2.00E-01	0.2	0.2	222	9.70
PCB 1254	11097-69-1	42800	2140000	6.94	0.02	4.10E-02	0.2	0.2	351	3.30
2-Nitroaniline	88-74-4	4.7	236	1.83	0.02	1.26E+03	0.2	0.2	1440	7.00
Methoxychlor	72-43-5	1600	80000	4.30	0.02	1.00E-01	0.2	0.2	non-volatile	199
Aldrin	309-00-2	1920	96000	3.01	0.02	1.80E-01	0.2	0.2	69.2	0.14
Beryllium	7440-41-7	0	0	0	0.02	0	0.2	0.2	non-volatile	0.77
Dieldrin	60-57-1	34.0	1700	3.50	0.02	1.95E-01	0.2	0.2	1.37	0.11
Heptachlor Epoxide	1024-57-3	4.4	220	2.70	0.02	3.50E-01	0.2	0.2	0.378	0.13

TABLE A4-8. Air Emission Rate Calculation Worksheet of Volatile and Semivolatile COPCs from Assumed Contaminated Subsurface Soil ($C_{\text{soil}} < C_{\text{sat}}$).

	CAS Number	C_{sat}	C_{soil}	A	D_e	D	ϵ	K_{a}	H (atm-m ³ /mol)	K_d	α	t	E	E
Production Area														
PCB 1248	12672-29-6	222	0.21	1.30E+08	0.0273	0.0550	0.120	2.96E-06	4.00E-04	5540	4.16E-09	9E+0	1.54E-07	1.18E-11
PCB 1254	11097-69-1	351	2.00	1.30E+08	0.0261	0.0525	0.120	1.92E-07	2.00E-04	4280	2.57E-10	9E+0	3.64E-07	2.80E-11
PCB 1260	11096-82-5	386	6.40	1.30E+08	0.0244	0.0491	0.120	7.65E-08	2.50E-04	1340	9.60E-11	9E+0	7.12E-07	5.48E-11
gamma-Chlordane	57-74-9	0.229	0.070	1.30E+08	0.0224	0.0450	0.120	1.04E-05	4.80E-05	190	1.19E-08	9E+0	8.68E-08	6.68E-12
Warwick Area														
PCB 1248	12672-29-6	222	9.70	3.25E+07	0.0273	0.0550	0.120	2.96E-06	4.00E-04	5540	4.16E-09	9E+0	1.78E-06	5.47E-10
PCB 1254	11097-69-1	351	3.30	3.25E+07	0.0261	0.0525	0.120	1.92E-07	2.00E-04	4280	2.57E-10	9E+0	1.50E-07	4.62E-11
2-Nitroaniline	88-74-4	1440	7.00	3.25E+07	0.0363	0.0730	0.120	4.35E-06	5.00E-07	4.72	8.11E-09	9E+0	1.79E-06	5.51E-10
Methoxychlor	72-43-5	nonvolatile	199	3.25E+07	0.0205	0.0412	0.120	nonvolatile	2.26E-08	1600		9E+0	nonvolatile	nonvolatile
Aldrin	309-00-2	69.2	0.14	3.25E+07	0.0236	0.0474	0.120	1.06E-05	4.96E-04	1920	1.28E-08	9E+0	4.51E-08	1.39E-11
Beryllium	7440-41-7	nonvolatile	0.77	3.25E+07	0	0	0.120	nonvolatile	0			9E+0	nonvolatile	nonvolatile
Dieldrin	60-57-1	1.37	0.11	3.25E+07	0.0242	0.0488	0.120	1.33E-05	1.10E-05	34.0	1.65E-08	9E+0	4.02E-08	1.24E-11
Heptachlor Epoxide	1024-57-3	0.378	0.13	3.25E+07	0.0228	0.0460	0.120	2.98E-04	3.20E-05	4.40	3.50E-07	9E+0	2.18E-07	6.72E-11

*NA denotes not available.

TABLE A4-9. Air Emission Rate Calculation Worksheet of Volatile, Semivolatile, and Nonvolatile COPCs from Wind Erodible Surface Soils.

Contaminant	CAS Number	V	u (m/s)	u _t (m/s)	F(x)	C _{soil} (mg/kg)	E ₁₀ (g/s-m ²)
Production Area							
PM-10	-	0.9	4.74	5.1	1.65	-	0
PCB 1248	12672-29-6	0.9	4.74	5.1	1.65	0.44	5.8e-13
PCB 1254	11097-69-1	0.9	4.74	5.1	1.65	3.6	4.8e-12
PCB 1260	11096-82-5	0.9	4.74	5.1	1.65	6.1	8.1e-12
gamma-Chlordane	57-74-9	0.9	4.74	5.1	1.65	0.13	1.7e-13
Warwick Area							
PM-10	-	0.9	4.74	5.1	1.65	-	0
PCB 1248	12672-29-6	0.9	4.74	5.1	1.65	15	2.0e-11
PCB 1254	11097-69-1	0.9	4.74	5.1	1.65	5.2	6.9e-12
2-Nitroaniline	88-74-4	0.9	4.74	5.1	1.65	7	9.3e-12
Methoxychlor	72-43-5	0.9	4.74	5.1	1.65	231	3.1e-10
Aldrin	309-00-2	0.9	4.74	5.1	1.65	0.21	2.8e-13
Beryllium	7440-41-7	0.9	4.74	5.1	1.65	0.72	9.7e-13
Dieldrin	60-57-1	0.9	4.74	5.1	1.65	0.16	2.1e-13
Heptachlor Epoxide	1024-57-3	0.9	4.74	5.1	1.65	0.19	2.5e-13

TABLE A4-10. Results of the Air Transport Analysis.

Contaminant	CAS Number	Predicted Air Concentration (µg/m³)		
		Gaseous Emissions from Subsurface Soils	Wind Blown Dust from Surface Soils	Ambient
Production Area				
PM-10	-	nonvolatile	3.90E+00	3.90E+00
PCB 1248	12672-29-6	3.48E-05	1.71E-06	3.65E-05
PCB 1254	11097-69-1	8.24E-05	1.40E-05	9.65E-05
PCB 1260	11096-82-5	1.61E-04	2.38E-05	1.85E-04
gamma-Chlordane	57-74-9	1.96E-05	5.07E-07	2.01E-05
Warwick Area				
PM-10	-	nonvolatile	3.56E+00	3.56E+00
PCB 1248	12672-29-6	1.47E-03	5.34E-05	1.52E-03
PCB 1254	11097-69-1	1.24E-04	1.85E-05	1.43E-04
2-Nitroaniline	88-74-4	1.48E-03	2.49E-05	1.51E-03
Methoxychlor	72-43-5	nonvolatile	8.23E-04	8.23E-04
Aldrin	309-00-2	3.73E-05	7.48E-07	3.80E-05
Beryllium	7440-41-7	nonvolatile	2.56E-06	2.56E-06
Dieldrin	60-57-1	3.32E-05	5.70E-07	3.38E-05
Heptachlor Epoxide	1024-57-3	1.81E-04	6.77E-07	1.81E-04

FIGURE A4-1. Area Source Configuration Used in the Air Dispersion Modeling.

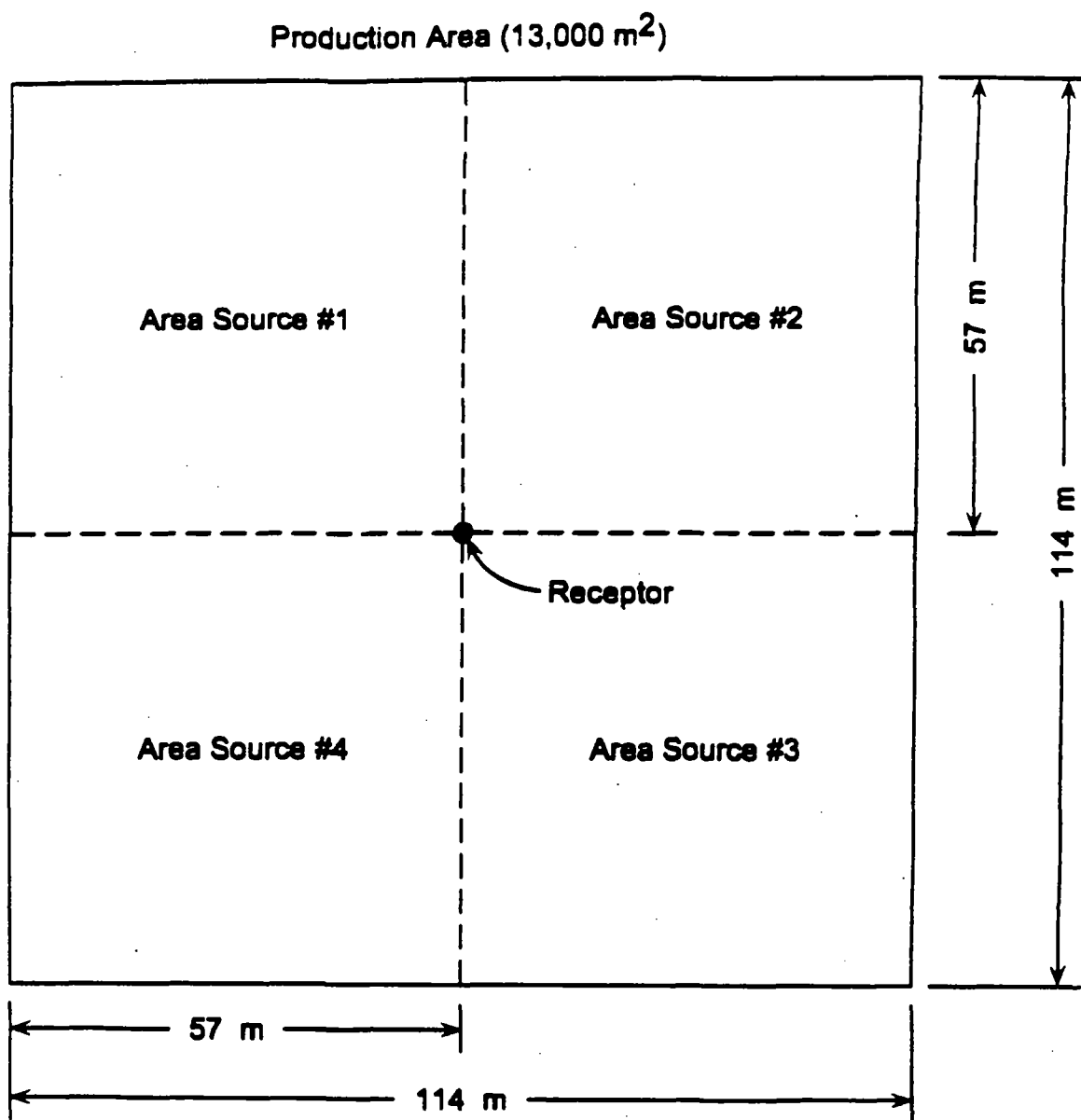


FIGURE A4-2. Gaseous Air Emissions From Contaminated Subsurface Soil.

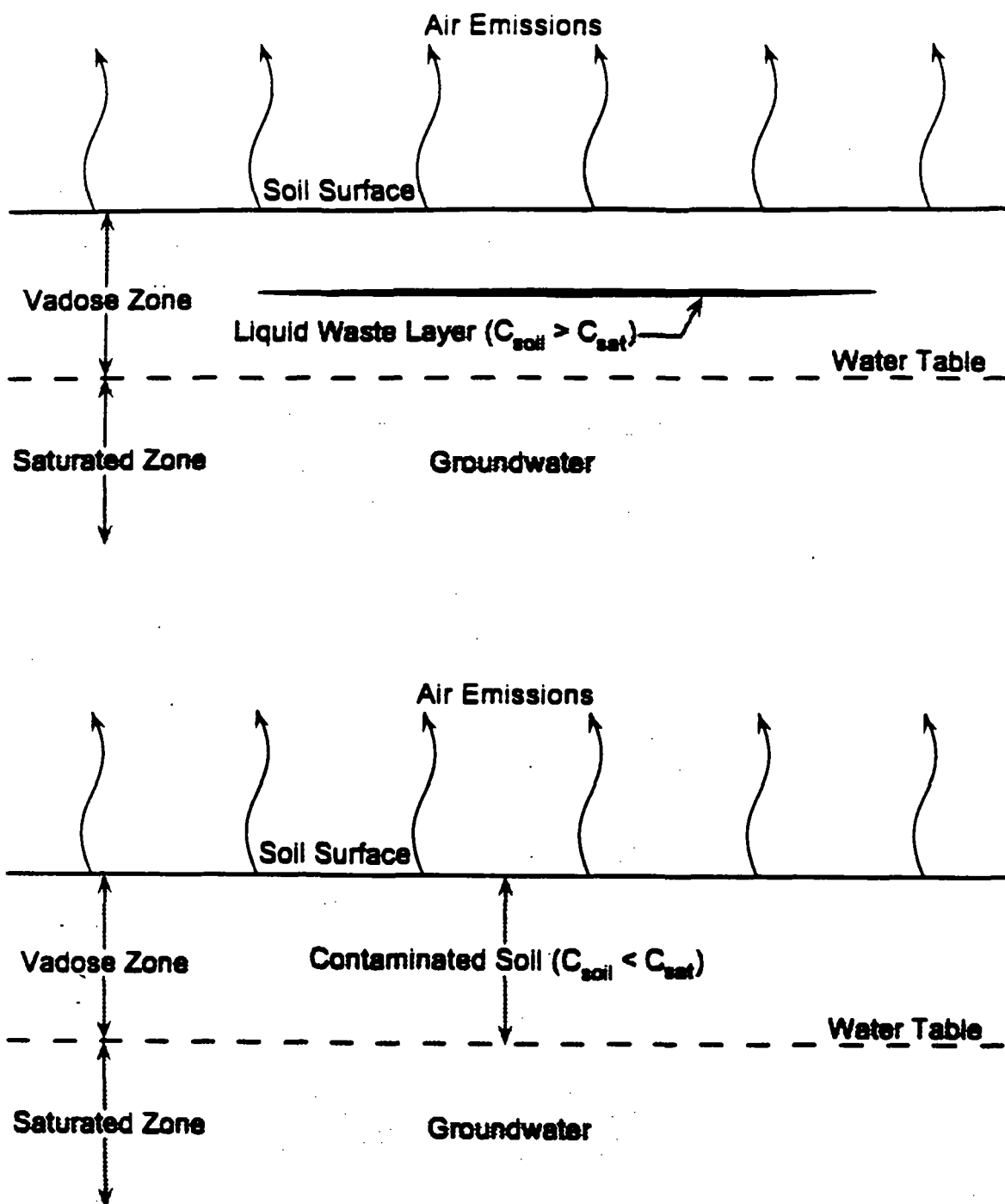


FIGURE A4-3. Soil Particle Size Distribution for the Production Area.

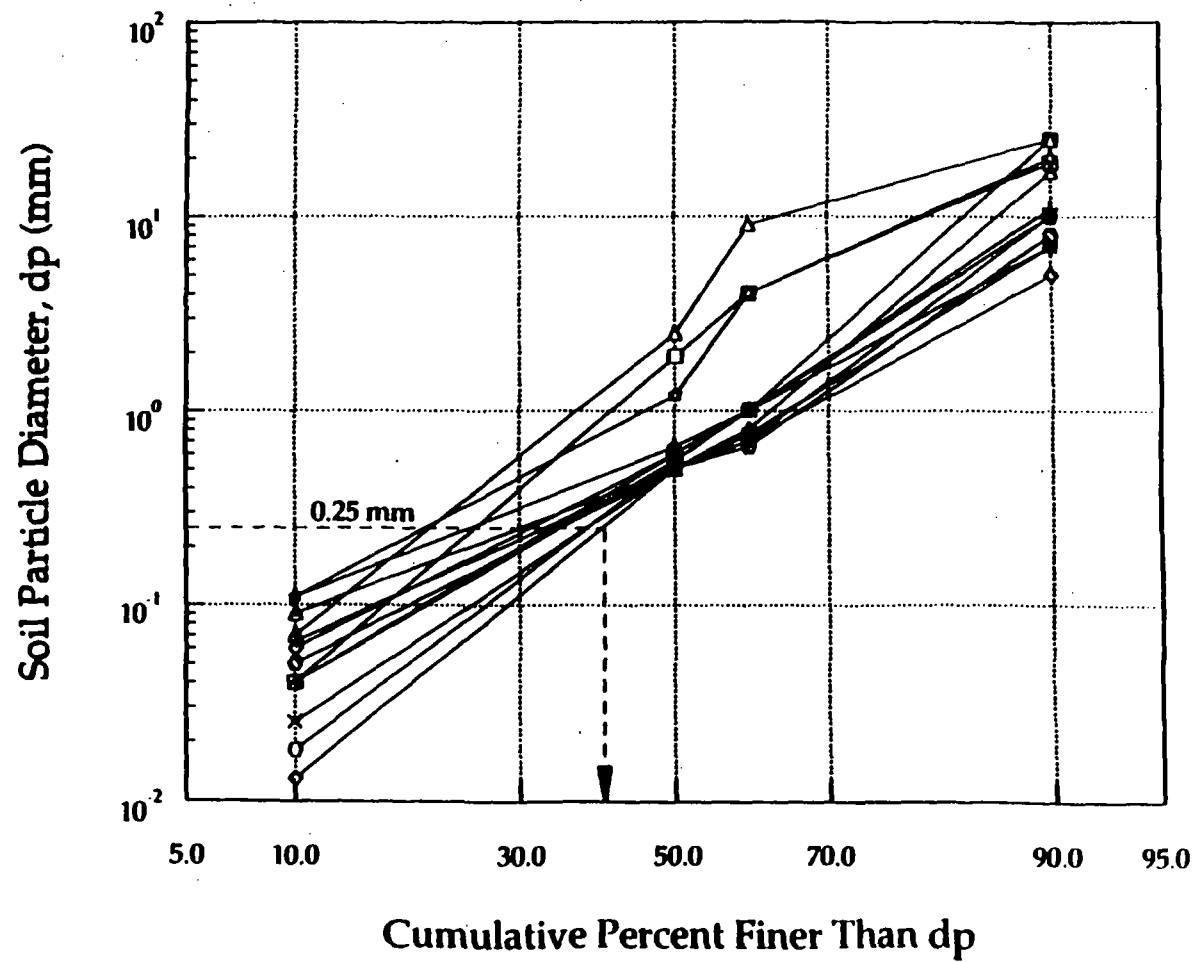


FIGURE A4-4. Soil Particle Size Distribution for the Warwick Area.

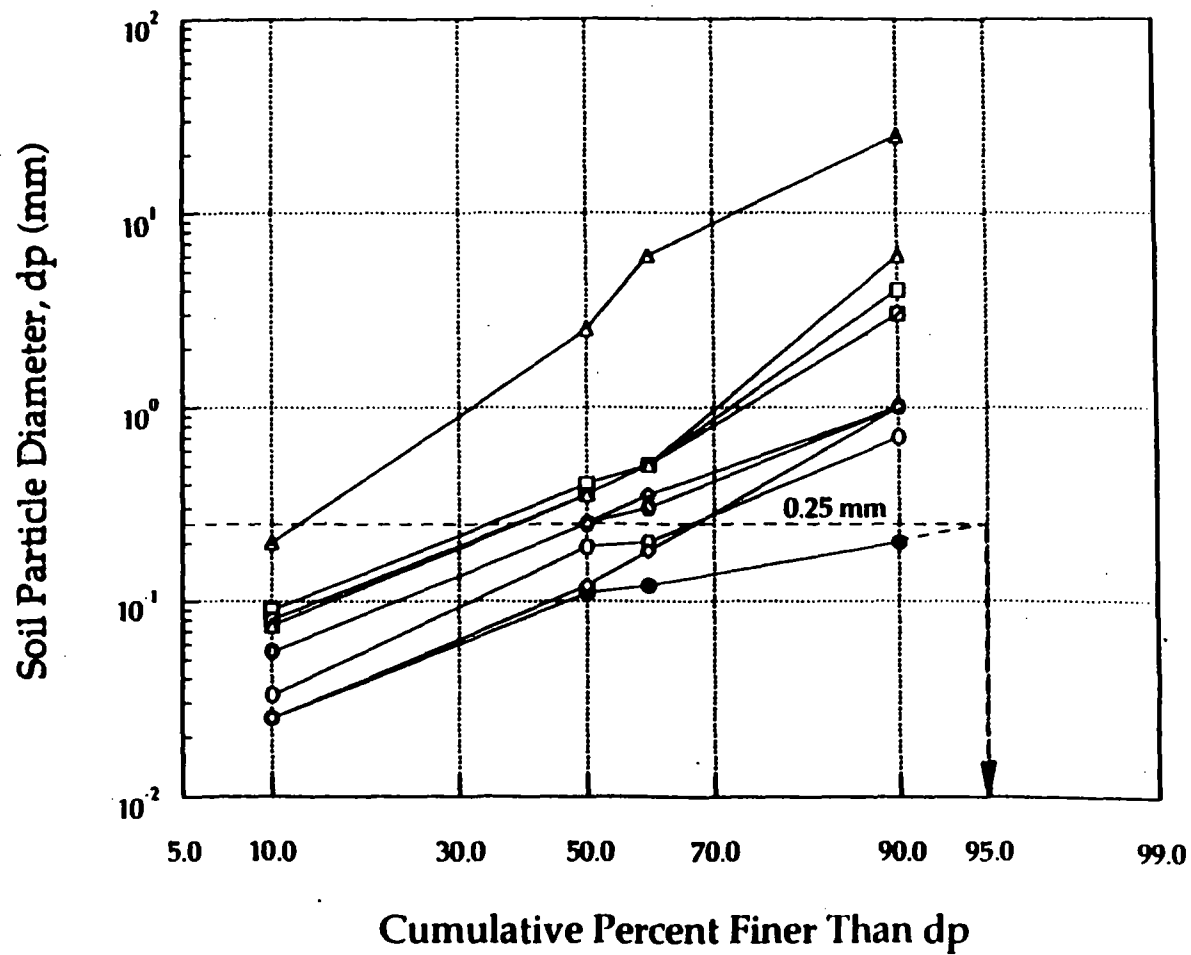


FIGURE A4-5. Threshold Friction Velocity Versus Aggregate Size Distribution.

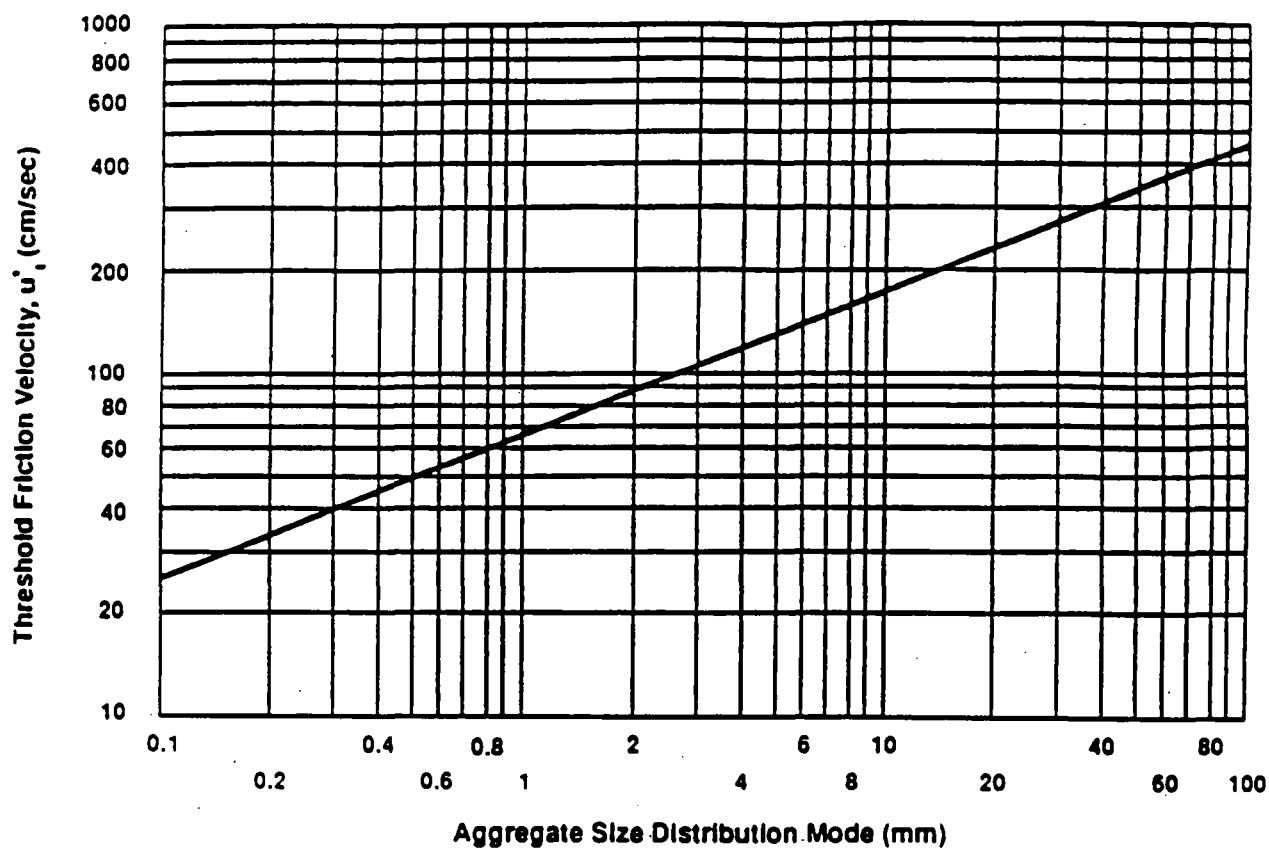
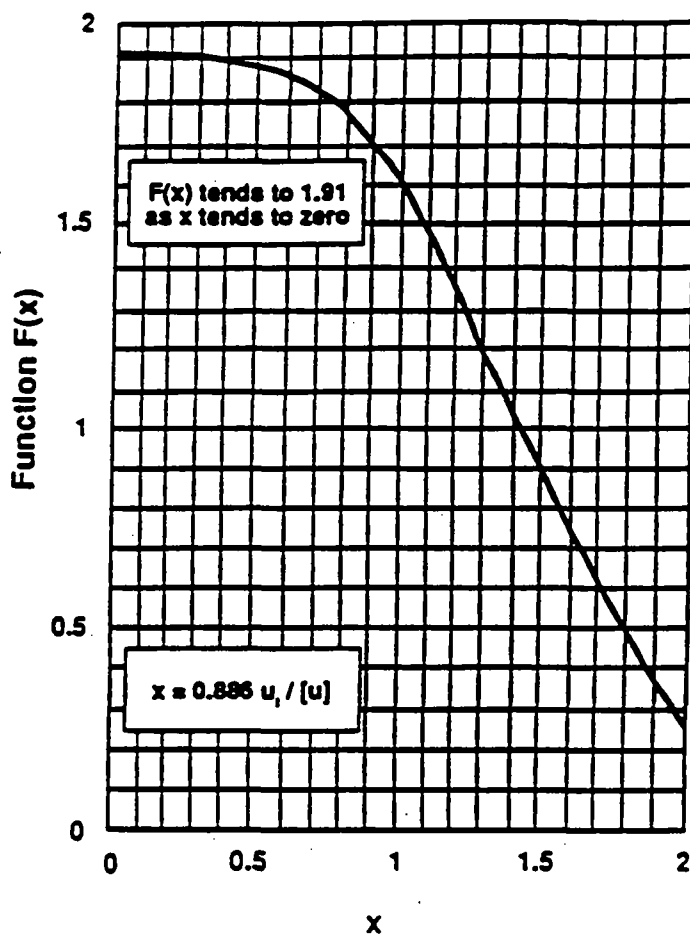


FIGURE A4-6. Function Curve Used in the Unlimited Reservoir Model.



NOTE: If $x > 2$,

$$F(x) = 0.18 (8x^3 + 12x) e^{-x^2}$$

ISCST2 Model Run Printout

```

SO STARTING
SO TITLEONE CIDA-DELTA CRANSTON RI State
SO MODELLOPT DFAULT CONC RURAL
SO AVERTIME 1
SO POLLUTED PM-10
SO RUNORNOT RUN
SO FINISHED

```

```

SO STARTING
**      ID      TYPE      X      Y      Z
**      (m)    (m)    (m)    (m)    (m)
**      .....
SO LOCATION PRODUCT1 AREA      0.      0.      0.
          PRODUCT2 AREA      0.     -56.9      0.
          PRODUCT3 AREA    -56.9     -56.9      0.
          PRODUCT4 AREA    -56.9      0.      0.

SO LOCATION WARNICK1 AREA      0.      0.      0.
          WARNICK2 AREA      0.     -28.5      0.
          WARNICK3 AREA    -28.5     -28.5      0.
          WARNICK4 AREA    -28.5      0.      0.

```

```

** Area Source      QS      HS      X0
** Parameters:      (g/s-m2) (m) (m)
**      .....
SO SRCPARAM PRODUCT1 0.00000100 0. 56.9
          PRODUCT2 0.00000100 0. 56.9
          PRODUCT3 0.00000100 0. 56.9
          PRODUCT4 0.00000100 0. 56.9

SO SRCPARAM WARNICK1 0.00000100 0. 28.5
          WARNICK2 0.00000100 0. 28.5
          WARNICK3 0.00000100 0. 28.5
          WARNICK4 0.00000100 0. 28.5

```

```

SO SRCGROUP PRODUCT PRODUCT1-PRODUCT4
          WARNICK WARNICK1-WARNICK4

```

SO FINISHED

```

RE STARTING
**      X      Y
**      (m)    (m)
**      .....
RE DISCCART      0.      0.
RE FINISHED

```

```

ME STARTING
ME INPUTFIL CRANSTON.MET
ME ANEMHGT 10.
ME SURFDATA 99999 1994 CRANSTON, RI
ME QAIRDATA 99999 1994 CRANSTON, RI
ME FINISHED

```

```

OU STARTING
OU RECTABLE ALLAVE FIRST
OU MAXTABLE ALLAVE 50
OU FINISHED

```

```

*****
*** SETUP Finishes Successfully ***
*****

```


*** MODELING OPTIONS USED: CONC RURAL FLAT DEFAULT

*** MODEL SETUP OPTIONS SUMMARY ***

**Model Is Setup For Calculation of Average CONCENTRATION Values.

**Model Uses RURAL Dispersion.

**Model Uses Regulatory DEFAULT Options:

1. Final Plume Rise.
2. Stack-tip Downwash.
3. Buoyancy-induced Dispersion.
4. Use Calms Processing Routine.
5. Not Use Missing Data Processing Routine.
6. Default Wind Profile Exponents.
7. Default Vertical Potential Temperature Gradients.
8. "Upper Bound" Values for Supersquat Buildings.
9. No Exponential Decay for RURAL Mode

**Model Assumes Receptors on FLAT Terrain.

**Model Assumes No FLAGPOLE Receptor Heights.

**Model Calculates 1 Short Term Average(s) of: 1-HR

**This Run Includes: 12 Source(s); 3 Source Group(s); and 1 Receptor(s)

**The Model Assumes A Pollutant Type of: PM-10

**Model Set To Continue RUNNING After the Setup Testing.

**Output Options Selected:

Model Outputs Tables of Highest Short Term Values by Receptor (RECTABLE Keyword)
Model Outputs Tables of Overall Maximum Short Term Values (MAXTABLE Keyword)

**NOTE: The Following Flags May Appear Following CONC Values: c for Calm Hours
m for Missing Hours
b for Both Calm and Missing Hours

**Misc. Inputs: Anem. Hgt. (m) = 10.00 ; Decay Coef. = 0.0000 ; Rot. Angle = 0.0
Emission Units = GRAMS/SEC ; Emission Rate Unit Factor = 0.10000E+07
Output Units = MICROGRAMS/M³

**Input Runstream File: CRANSTON.DAT

; **Output Print File: CRANSTON.OUT

*** ISSUES VERSION 9103 *** *** DATA CHECK: CLEARED RI Site ***

*** MODELLING OPTIONS USED: CONC RURAL FLAT DFACET

*** AREA SOURCE DATA ***

SOURCE ID	NUMBER PART. CATS.	EMISSION RATE (GRAMS/SEC (METER**2))	COORD (SW CORNER X Y (METERS) (METERS)	BASE ELEV. (METERS)	RELEASE HEIGHT (METERS)	WIDTH OF AREA (METERS)	EMISSION RATE SCALAR VARY BY
PRODUCT1	1	0.10000E-05	0.0 0.0	0.0	0.00	56.90	
PRODUCT2	0	0.10000E-05	0.0 -56.9	0.0	0.00	56.90	
PRODUCT3	0	0.10000E-05	-56.9 -56.9	0.0	0.00	56.90	
PRODUCT4	0	0.10000E-05	-56.9 0.0	0.0	0.00	56.90	
WARWICK1	0	0.10000E-05	0.0 0.0	0.0	0.00	28.50	
WARWICK2	0	0.10000E-05	0.0 -28.5	0.0	0.00	28.50	
WARWICK3	0	0.10000E-05	-28.5 -28.5	0.0	0.00	28.50	
WARWICK4	0	0.10000E-05	-28.5 0.0	0.0	0.00	28.50	

*** ISOSTE VERSION 10119 *** *** DATA-BASE OPERATOR RI 10119 ***

PAGE

*** MODELING OPTIONS USED: CONC RURAL FLAT DEFACET

*** SOURCE IDs DEFINING SOURCE GROUPS ***

GROUP ID

SOURCE ID:

PRODUCT PRODUCT1, PRODUCT2, PRODUCT3, PRODUCT4,

WARWICK WARWICK1, WARWICK2, WARWICK3, WARWICK4,

*** ISSUES VERSION 1.000 *** *** Data: Data: Data: AD Site

*** MODELING OPTIONS USED: CONC RURAL FLAT DEFAULT

*** DISCRETE CARTESIAN RECEPTORS ***
X-COORD Y-COORD Z-LEVEL Z-FLAG
(METERS)

0.0 0.0 0.0 0.01.....

PAGE

YES: 0=NC

[illegible]

NOTE: METEOROLOGICAL DATA ACTUALLY PROCESSED WILL ALSO DEPEND ON WHAT IS INCLUDED IN THE DATA FILE.

(METERS / SEC)

1.54. 3.09. 5.14. 8.23. 10.80.

*** WIND PROFILE EXPONENTS ***

STABILITY CATEGORY	WIND SPEED CATEGORY					
	1	2	3	4	5	6
A	.70000E-01	.70000E-01	.70000E-01	.70000E-01	.70000E-01	.70000E-01
B	.70000E-01	.70000E-01	.70000E-01	.70000E-01	.70000E-01	.70000E-01
C	.10000E+00	.10000E+00	.10000E+00	.10000E+00	.10000E+00	.10000E+00
D	.15000E+00	.15000E+00	.15000E+00	.15000E+00	.15000E+00	.15000E+00
E	.35000E+00	.35000E+00	.35000E+00	.35000E+00	.35000E+00	.35000E+00
F	.55000E+00	.55000E+00	.55000E+00	.55000E+00	.55000E+00	.55000E+00

(DEGREES KELVIN PER METER)

STABILITY CATEGORY	WIND SPEED CATEGORY					
	1	2	3	4	5	6
A	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
B	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
C	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
D	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
E	.20000E-01	.20000E-01	.20000E-01	.20000E-01	.20000E-01	.20000E-01
F	.35000E-01	.35000E-01	.35000E-01	.35000E-01	.35000E-01	.35000E-01

*** INPUT VERSION FILE *** *** DATA DATE: 01/01/95 ***

*** MODELING OPTIONS USED: CONC RURAL FLAT DELETED

*** THE FIRST 6 HOURS OF METEOROLOGICAL DATA ***

FILE: CRANSTON.MET

FORMAT: 400.2F5.4 F0.1.00.2F1.1

SURFACE STATION NO.: 99999

UPPER AIR STATION NO.: 99999

NAME: CRANSTON

NAME: CRANSTON

YEAR: 1994

YEAR: 1994

YEAR	MONTH	DAY	HOOR	FLOW VECTOR	SPEED (M/S)	TEMP (K)	STAB CLASS	MIXING HEIGHT (M) RURAL URBAN
94	1	1	1	360.0	4.74	283.0	4	1520.0 1520.0
94	1	1	2	10.0	4.74	283.0	4	1520.0 1520.0
94	1	1	3	20.0	4.74	283.0	4	1520.0 1520.0
94	1	1	4	30.0	4.74	283.0	4	1520.0 1520.0
94	1	1	5	40.0	4.74	283.0	4	1520.0 1520.0
94	1	1	6	45.0	4.74	283.0	4	1520.0 1520.0

*** NOTES: STABILITY CLASS 1=A, 2=B, 3=C, 4=D, 5=E AND 6=F.
FLOW VECTOR IS DIRECTION TOWARD WHICH WIND IS BLOWING.

*** MODELING OPTIONS USED: CONC RURAL FLAT DEFAULT

*** THE 1ST HIGHEST 1-HR AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP PRODUCT ***
INCLUDING SOURCES: PRODUCT1 PRODUCT2 PRODUCT3 PRODUCT4

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF PM-10 IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	(YYMMDDHH)	X-COORD (M)	Y-COORD (M)	CONC	(YYMMDDHH)
0.00	0.00	2.94176	(94010102)				

... MODELING OPTIONS USED CONC RURAL FLAT DEFAULT

*** THE 1ST HIGHEST 1-HR AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP WARRICK ***
 INCLUDING SOURCES WARRICK WARRICK WARRICK WARRICK

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF PM-10 IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC (YYMMDDHH)	X-COORD (M)	Y-COORD (M)	CONC (YYMMDDHH)
0.00	0.00	2.68840 (94010102)			

*** MODELING OPTIONS USED: CONC RURAL FLAT DELETED

*** THE MAXIMUM 50 L-HR AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP PRODUCT ***
INCLUDING SOURCES: PRODUCT1 PRODUCT2 PRODUCT3 PRODUCT4

*** CONC OF PM-10 IN MICROGRAMS/M**3 ***

RANK	CONC	(YMMDDHH) AT	RECEPTOR (XR.YR. OF TYPE	RANK	CONC	(YMMDDHH) AT	RECEPTOR (XR.YR. OF TYPE
1.	2.94176	(94010102) AT	0.00. 0.00: DC	26.	0.00000	() AT	0.00. 0.00:
2.	2.79475	(94010103) AT	0.00. 0.00: DC	27.	0.00000	() AT	0.00. 0.00:
3.	2.70293	(94010104) AT	0.00. 0.00: DC	28.	0.00000	() AT	0.00. 0.00:
4.	2.65875	(94010105) AT	0.00. 0.00: DC	29.	0.00000	() AT	0.00. 0.00:
5.	2.65331	(94010106) AT	0.00. 0.00: DC	30.	0.00000	() AT	0.00. 0.00:
6.	0.00000	() AT	0.00. 0.00:	31.	0.00000	() AT	0.00. 0.00:
7.	0.00000	() AT	0.00. 0.00:	32.	0.00000	() AT	0.00. 0.00:
8.	0.00000	() AT	0.00. 0.00:	33.	0.00000	() AT	0.00. 0.00:
9.	0.00000	() AT	0.00. 0.00:	34.	0.00000	() AT	0.00. 0.00:
10.	0.00000	() AT	0.00. 0.00:	35.	0.00000	() AT	0.00. 0.00:
11.	0.00000	() AT	0.00. 0.00:	36.	0.00000	() AT	0.00. 0.00:
12.	0.00000	() AT	0.00. 0.00:	37.	0.00000	() AT	0.00. 0.00:
13.	0.00000	() AT	0.00. 0.00:	38.	0.00000	() AT	0.00. 0.00:
14.	0.00000	() AT	0.00. 0.00:	39.	0.00000	() AT	0.00. 0.00:
15.	0.00000	() AT	0.00. 0.00:	40.	0.00000	() AT	0.00. 0.00:
16.	0.00000	() AT	0.00. 0.00:	41.	0.00000	() AT	0.00. 0.00:
17.	0.00000	() AT	0.00. 0.00:	42.	0.00000	() AT	0.00. 0.00:
18.	0.00000	() AT	0.00. 0.00:	43.	0.00000	() AT	0.00. 0.00:
19.	0.00000	() AT	0.00. 0.00:	44.	0.00000	() AT	0.00. 0.00:
20.	0.00000	() AT	0.00. 0.00:	45.	0.00000	() AT	0.00. 0.00:
21.	0.00000	() AT	0.00. 0.00:	46.	0.00000	() AT	0.00. 0.00:
22.	0.00000	() AT	0.00. 0.00:	47.	0.00000	() AT	0.00. 0.00:
23.	0.00000	() AT	0.00. 0.00:	48.	0.00000	() AT	0.00. 0.00:
24.	0.00000	() AT	0.00. 0.00:	49.	0.00000	() AT	0.00. 0.00:
25.	0.00000	() AT	0.00. 0.00:	50.	0.00000	() AT	0.00. 0.00:

*** RECEPTOR TYPES: GC = GRIDCART
GP = GRIDPOLA
DC = DISCCART
DP = DISCPOLA
BD = BOUNDARY

*** MODELING OPTIONS USE CONC RURAL PLAT SPATIAL

*** THE MAXIMUM 51 1-HR AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: WARWICK ***
INCLUDING SOURCE:5 WARWICK WARWICK WARWICK WARWICK

** CONC OF PM-10 IN MICROGRAMS/M**3

RANK	CONC	YYMMDDHH	AT	RECEPTOR (XR.YR) OF TYPE	RANK	CONC	YYMMDDHH	AT	RECEPTOR (XR.YR) OF TYPE
1.	2.68840	194010102	AT	0.00, 0.00) DC	26.	0.00000	01 AT	0.00, 0.00	
2.	2.55405	194010103	AT	0.00, 0.00) DC	27.	0.00000	01 AT	0.00, 0.00	
3.	2.47014	194010104	AT	0.00, 0.00) DC	28.	0.00000	01 AT	0.00, 0.00	
4.	2.42977	194010105	AT	0.00, 0.00) DC	29.	0.00000	01 AT	0.00, 0.00	
5.	2.42479	194010106	AT	0.00, 0.00) DC	30.	0.00000	01 AT	0.00, 0.00	
6.	0.00000	01 AT		0.00, 0.00)	31.	0.00000	01 AT	0.00, 0.00	
7.	0.00000	01 AT		0.00, 0.00)	32.	0.00000	01 AT	0.00, 0.00	
8.	0.00000	01 AT		0.00, 0.00)	33.	0.00000	01 AT	0.00, 0.00	
9.	0.00000	01 AT		0.00, 0.00)	34.	0.00000	01 AT	0.00, 0.00	
10.	0.00000	01 AT		0.00, 0.00)	35.	0.00000	01 AT	0.00, 0.00	
11.	0.00000	01 AT		0.00, 0.00)	36.	0.00000	01 AT	0.00, 0.00	
12.	0.00000	01 AT		0.00, 0.00)	37.	0.00000	01 AT	0.00, 0.00	
13.	0.00000	01 AT		0.00, 0.00)	38.	0.00000	01 AT	0.00, 0.00	
14.	0.00000	01 AT		0.00, 0.00)	39.	0.00000	01 AT	0.00, 0.00	
15.	0.00000	01 AT		0.00, 0.00)	40.	0.00000	01 AT	0.00, 0.00	
16.	0.00000	01 AT		0.00, 0.00)	41.	0.00000	01 AT	0.00, 0.00	
17.	0.00000	01 AT		0.00, 0.00)	42.	0.00000	01 AT	0.00, 0.00	
18.	0.00000	01 AT		0.00, 0.00)	43.	0.00000	01 AT	0.00, 0.00	
19.	0.00000	01 AT		0.00, 0.00)	44.	0.00000	01 AT	0.00, 0.00	
20.	0.00000	01 AT		0.00, 0.00)	45.	0.00000	01 AT	0.00, 0.00	
21.	0.00000	01 AT		0.00, 0.00)	46.	0.00000	01 AT	0.00, 0.00	
22.	0.00000	01 AT		0.00, 0.00)	47.	0.00000	01 AT	0.00, 0.00	
23.	0.00000	01 AT		0.00, 0.00)	48.	0.00000	01 AT	0.00, 0.00	
24.	0.00000	01 AT		0.00, 0.00)	49.	0.00000	01 AT	0.00, 0.00	
25.	0.00000	01 AT		0.00, 0.00)	50.	0.00000	01 AT	0.00, 0.00	

*** RECEPTOR TYPES: GC = GRIDCART
GP = GRIDPOLR
DC = DISCCART
DP = DISCPOLR
BD = BOUNDARY

*** ISCST3 VERSION 95109 *** *** Ciba-Geigy, Cranston, RI Site ***

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*** MODELING OPTIONS USED: CONC RURAL PLAT DPAUSE

*** THE SUMMARY OF HIGHEST 1-HR RESULTS ***

** CONC OF PM-10 IN MICROGRAMS/M**3 **

GROUP ID	AVERAGE CONC	DATE (YYMMDDHH)	RECEPTOR	(XR. YR.	ZELEV.	ZFLAG.	OF TYPE	NETWORK GRID-ID
PRODUCT HIGH 1ST HIGH VALUE IS	2.94176	ON 94010102: AT (0.00.	0.00.	0.00.	0.00.	DC	
WARWICK HIGH 1ST HIGH VALUE IS	2.68840	ON 94010102: AT (0.00.	0.00.	0.00.	0.00.	DC	

*** RECEPTOR TYPES: GC = GRIDCART
GP = GRIDPOLR
DC = DISCCART
DP = DISCPOLR
BD = BOUNDARY

*** ISCST2 VERSION 91129 *** *** Ciba-Geigy Cranston RI Site ***

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*** MODELING OPTIONS USED: CONC RURAL FLAT OFAC10

*** Message Summary For ISC2 Model Execution ***

***** Summary of Total Messages *****

A Total of 0 Fatal Error Message(s)
A Total of 0 Warning Message(s)
A Total of 0 Informational Message(s)

***** FATAL ERROR MESSAGES *****
*** NONE ***

***** WARNING MESSAGES *****
*** NONE ***

*** ISCST2 Finishes Successfully ***

Attachment 5

IRIS Toxicity Printouts

PCB 1254

1 - IRIS
 NAME - Aroclor 1254
 RN - 11097-69-1
 IRSN - 662
 DATE - 941003
 UPDT - 10/03/94, 5 fields
 STAT - Oral RfD Assessment (RDO) on-line 10/01/94
 STAT - Inhalation RfC Assessment (RDI) no data
 STAT - Carcinogenicity Assessment (CAR) no data
 STAT - Drinking Water Health Advisories (DWHA) no data
 STAT - U.S. EPA Regulatory Actions (EXSR) no data
 STAT - Supplementary Data no data
 IRH - 07/01/93 RDO Oral RfD now under review
 IRH - 03/01/94 RDO Work group review date added
 IRH - 10/01/94 RDO Oral RfD summary on-line
 IRH - 10/01/94 OREF Oral RfD references on-line
 RLEN - 63965
 SY - Aroclor 1254
 SY - Arochlor 1254
 SY - CHLORIERTE BIPHENYLE, CHLORGEHALT 54% [German]
 SY - CLORODIFENILI, CLORO 54% [Italian]
 SY - DIPHENYLE CHLORE, 54% DE CHLORE [French]
 SY - HSDB 6357
 SY - NCI-C02664

RDO -

o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	NOAEL: None LOAEL: 0.005 mg/kg-day	300	1	2E-5 mg/kg-day

Monkey Clinical and Immunologic Studies

Arnold et al., 1994a,b;
 Tryphonas et al., 1989,
 1991a,b

*Conversion Factors and Assumptions – None

o ORAL RFD STUDIES :

Arnold, D.L., F. Bryce, R. Stapley et al. 1993a. Toxicological consequences of Aroclor 1254 ingestion by female Rhesus (*Macaca mulatta*) monkeys, Part 1A: Prebreeding phase - clinical health findings. Food Chem. Toxicol. 31: 799-810.

Arnold, D.L., F. Bryce, K. Karpinski et al. 1993b. Toxicological consequences of Aroclor 1254 ingestion by female Rhesus (*Macaca mulatta*) monkeys, Part 1B: Prebreeding phase -clinical and analytical laboratory findings. *Food Chem. Toxicol.* 31: 811-824.

Tryphonas, H., S. Hayward, L. O'Grady et al. 1989. Immunotoxicity studies of PCB (Aroclor 1254) in the adult rhesus (*Macaca mulatta*) monkey - preliminary report. *Int. J. Immunopharmacol.* 11: 199-206.

Tryphonas, H., M.I. Luster, G. Schiffman et al. 1991a. Effect of chronic exposure of PCB (Aroclor 1254) on specific and nonspecific immune parameters in the rhesus (*Macaca mulatta*) monkey. *Fund. Appl. Toxicol.* 16(4): 773-786.

Tryphonas, H., M.I. Luster, K.L. White et al. 1991b. Effects of PCB (Aroclor 1254) on non-specific immune parameters in Rhesus (*Macaca mulatta*) monkeys. *Int. J. Immunopharmacol.* 13: 639-648.

Groups of 16 adult female rhesus monkeys ingested gelatin capsules containing Aroclor 1254 (Monsanto Lot No. KA634) in 1:1 glycerol: corn oil vehicle daily at dosages of 0, 5, 20, 40 or 80 ug/kg-day for more than 5 years. The Aroclor mixture contained 5.19 ppm of polychlorinated dibenzofurans and undetectable levels of polychlorinated dibenzo-p-dioxins (Truelove et al., 1990). At study initiation the monkeys were 11.1 +/- 4.1 years old (Tryphonas et al., 1991a,b; Arnold et al., 1993a,b). After 25 months of exposure the monkeys had achieved a pharmacokinetic steady-state based on PCB concentrations in adipose tissue and/or blood (Tryphonas et al., 1989). Results of general health and clinical pathology evaluations conducted during the first 37 months of exposure were reported by Arnold et al. (1993a,b). Results of immunologic assessments after 23 and 55 months of exposure were reported by Tryphonas et al. (1989, 1991a,b). Results of reproductive endocrinology evaluations after 24 or 29 months of exposure were reported by Truelove et al. (1990) and Arnold et al. (1993a). Effects on hydrocortisone levels during the first 22 months of exposure were reported by Loo et al. (1989) and Arnold et al. (1993b). All of the aforementioned evaluations were performed during the prebreeding phase of the study. Results of reproduction and histopathology evaluations in these monkeys are not fully available (Arnold, 1992).

General health status was evaluated daily, and body weight measurements, feed conversion ratio calculations, and detailed clinical evaluations were performed weekly throughout the study. Analyses of clinical signs of toxicity were limited to the occurrence of eye exudate, inflammation and/or prominence of the eyelid Meibomian (tarsal) glands, and particular changes in finger and toe nails (prominent nail beds, separation from nail beds, elevated nail beds, and nails folding on themselves). Each endpoint was analyzed for individual treatment-control group differences and dose-related trends with respect to incidence rate, total frequency of observed occurrences, and the onset time of the condition. With respect to effects on the eyes, the treatment-control group comparisons showed statistically significant (p less than or equal to 0.05) increases in the total frequency of inflamed and/or prominent Meibomian glands at 0.005, 0.02 and 0.08 mg/kg-day, and decreased onset time for these effects at 0.08 mg/kg-day. Significant dose-related trends (p less than or equal to 0.05) were observed for increased total frequencies of inflamed and/or prominent Meibomian glands, decreased onset time of inflamed and/or

prominent Meibomian glands, and increased incidences of eye exudate. With respect to effects on finger and/or toe nails, the treatment-control group comparisons showed significantly (p less than or equal to 0.05) increased incidence of certain nail changes at 0.005 mg/kg-day (nail folding) and 0.08 mg/kg-day (elevated nails), increased total frequency of certain nail changes at 0.005 mg/kg-day (nail separation), 0.04 mg/kg-day (nail folding and separation) and 0.08 mg/kg-day (nail folding and separation, prominent beds, elevated nails), and decreased onset time of certain nail changes at 0.005 mg/kg-day (elevated nails) and 0.08 mg/kg-day (nail folding, prominent beds, elevated nails). Significant dose-related trends (p less than or equal to 0.05) were observed for certain nail changes (prominent beds, elevated nails) when adjusted for onset time, total frequencies of certain nail changes (nail folding and separation, prominent beds, elevated nails), and decreases in onset time of certain nail changes (nail folding, prominent beds, elevated nails).

Immunologic assessment showed significant ($p < 0.01$ or < 0.05) reductions in IgG (at all doses of Aroclor 1254) and IgM (all doses but 0.02 mg/kg-day) antibody levels in response to injected sheep red blood cells (SRBC) after 23 months of exposure (Tryphonas et al., 1989). A significant ($p < 0.05$) decrease in the percent of helper T-lymphocytes, a significant ($p < 0.05$) increase in the percent and absolute level of suppressor T-lymphocytes (TS) and a significant ($p < 0.01$) reduction in TH/TS ratio was observed at 0.08 mg/kg-day. The antibody response to SRBC is an antigen-driven response that requires the interaction of several distinct cell types (i.e., antigen processing and presentation by macrophages, participation by T-helper cells and finally proliferation and differentiation of B cells into plasma cells that secrete the antibody), which result in the production and secretion of antibodies specific for SRBC from plasma cells. Perturbation in any of the cells or cell-to-cell interactions by physical, chemical or biological agents can result in aberrant antibody responses. The necessity for the interaction of the three principal cells of the immune system (i.e., macrophage, B lymphocyte and T lymphocyte), in response to SRBC, is the main reason why this response has been so widely used in immunotoxicity testing as a surrogate for infection with a pathogenic organism.

In a recent evaluation of the sensitivity and predictability of various immune function assays used for immunotoxicity testing in the mouse (Luster et al., 1992), the antibody plaque-forming cell (PFC) response to SRBC was found to show the highest association with immunotoxic compounds. Essentially this means that the antibody PFC response to SRBC is a very good predictor of immunotoxicants. Also, it has recently been demonstrated that measurement of serum antibody titer to SRBC using the ELISA assay is as sensitive as the PFC assay for determining the response to SRBC (Butterworth et al., 1993).

There were no exposure-related effects on total B-lymphocytes, total T-lymphocytes, total serum immunoglobulin levels, total serum protein, serum protein fractions after 23 months. No exposure-related effects on serum hydrocortisone levels were observed although the SRBC assay is considered a good surrogate (Tryphonas et al., 1989; Loo et al., 1989; Arnold et al., 1993b).

After 55 months of exposure, there was a significant dose-related decrease ($p < 0.0005$ for pairwise comparisons and trend test) in the IgM antibody

response to injected SRBC at greater than or equal to 0.005 mg/kg-day at all times of evaluation (1-4 weeks postimmunization) (Tryphonas et al., 1991a). IgG antibody response to injected SRBC was significantly ($p < 0.01$) decreased only at 0.04 mg/kg-day, although the overall trend for dose-response was significant ($p = 0.033$). The antibody response to pneumococcus antigen did not differ significantly among all test groups (including controls) at any time tested and showed no dose-related trend. However, the antibody response to pneumococcus antigen is a T cell-independent response and the fact that there is no change with this antigen is not inconsistent with the depressed response to the T cell-dependent SRBC antigen. Other data corroborate the significance of Aroclor 1254 suppression of the antibody response to SRBC and point to effects on T lymphocytes including the dose-related suppression of the Con A and PHA lymphoproliferative responses. The monkeys treated with greater than or equal to 0.005 mg/kg-day had significantly ($p < 0.0001$) lower mean percentage levels of total T-lymphocytes and significant trend for dose-response, but absolute numbers of T-lymphocytes were similar among test groups. Flow cytometric analysis showed no treatment-related effects on peripheral blood T-helper, T-suppressor or B-lymphocytes or TH/TS lymphocyte ratio. A statistically significant, dose-related increase was noted for thymosin alpha-1-levels but not for thymosin beta-2-levels. Serum complement activity was significantly ($p < 0.025$) increased at greater than or equal to 0.005 mg/kg-day but showed no significant ($p = 0.1$) dose-related trend. Natural killer cell activity at effect or target ratios of 25:1, 50:1 or 75:1 was not significantly ($p > 0.05$) increased at any dosage, although there was a significant ($p = 0.03$) dose-related trend. No signs of microbial infection were noted in any of the preceding reports.

Clinical pathology was evaluated during the first 37 months of the study (Arnold et al., 1993b). These evaluations included monthly measurements of hematology and serum biochemistry (including serum protein, RBC indices, semi-monthly measurements of thyroid function, and daily measurements of urinary porphyrins during the 33rd month of dosing). Significant ($p < 0.05$) decreases in average dose-group values compared with controls were found for serum cholesterol at 0.04 mg/kg-day, and reticulocyte count, serum cholesterol, total bilirubin, and alpha-1 + alpha-2-globulins at 0.08 mg/kg-day. Significant dose-related decreasing linear trends were also observed for reticulocyte count ($p = 0.002$), cholesterol (p less than or equal to 0.001), and total bilirubin ($p = 0.005$). Dose-related decreasing linear trends were also observed for red blood cell count ($p = 0.019$), mean platelet volume ($p = 0.034$), hematocrit ($p = 0.064$), hemoglobin concentration ($p = 0.041$). With regard to thyroid endpoints [serum thyroxine (T4), serum triiodothyronine (T3) uptake ratio, percent T3 uptake, and free thyroxine index], dose-response analysis consisted of group mean comparisons and an assessment of parallelism in the response profiles (an absence of parallelism would indicate time-dose interactive effects). No statistically significant changes were observed for any of the thyroid endpoints.

After approximately 2 years of dosing, each dose group was randomly divided into two test groups for daily analyses of serum progesterone and estrogen concentrations during one menstrual cycle (Truelove et al., 1990; Arnold et al., 1993b). There were no statistically significant differences between treated and control monkeys in menstrual cycle length or menses duration, and no apparent treatment-related effects on incidence of anovulatory cycles or temporal relationship between estrogen peak and menses

onset, menses end or progesterone peak (Truelove et al., 1990; Arnold et al., 1993a,b).

To summarize the above, monkeys that ingested 0.005-0.08 mg/kg-day doses of Aroclor 1254 showed ocular exudate, prominence and inflammation of the Meibomian glands and distortion in nail bed formation. These changes were seen at the lowest dose tested, 0.005 mg/kg-day, and a dose-dependent response was demonstrated. Similar changes have been documented in humans for accidental oral ingestion of PCBs. Among the various immunologic function tests that were performed, the increases in IgM and IgG antibodies to sheep erythrocytes are most significant. IgG and IgM antibodies in response to SRBC were reduced after 23 months of exposure but only the IgM antibodies were clearly decreased after 55 months. Particular importance is attributed to the immune response to sheep erythrocytes since it involves participation by the three principal cells of the immune system: the macrophage, B lymphocytes and T lymphocytes and has been shown to be the most predictive immunotoxicity test of those currently in use (Luster et al., 1992). On the basis the studies described, a LOAEL of 0.005 mg/kg-day was established for Aroclor 1254.

o ORAL RFD UNCERTAINTY :

UF -- A 10-fold factor is applied to account for sensitive individuals. A factor of 3 is applied to extrapolation from rhesus monkeys to humans. A full 10-fold factor for interspecies extrapolation is not considered necessary because of similarities in toxic responses and metabolism of PCBs between monkeys and humans and the general physiologic similarity between these species. A partial factor is applied for the use of a minimal LOAEL since the changes in the periocular tissues and nail bed seen at the 0.05 mg/kg-day are not considered to be of marked severity. The duration of the critical study continued for approximately 25% of the lifespan of rhesus monkeys so that a reduced factor was used for extrapolation from subchronic exposure to a chronic RfD. The immunologic and clinical changes that were observed did not appear to be dependent upon duration which further justifies using a factor of 3 rather than 10 for extrapolation from subchronic to chronic, lifetime exposure. The total UF is 300.

o ORAL RFD MODIFYING FACTOR :

MF -- None

o ORAL RFD COMMENTS :

Human data available for risk assessment of Aroclor 1254 are useful only in a qualitative manner. Studies of the general population who were exposed to PCBs by consumption of contaminated food, particularly neurobehavioral evaluations of infants exposed in utero and/or through lactation, have been reported, but the original PCB mixtures, exposure levels and other details of exposure are not known (Kreiss et al., 1981; Humphrey, 1983; Fein et al., 1984a,b; Jacobson et al., 1984a, 1985, 1990a,b; Rogan et al., 1986; Gladen et al., 1988). Most of the information on health effects of PCB mixtures in humans is available from studies of occupational exposure. Some of these studies examined workers who had some occupational exposure to Aroclor 1254, but sequential or concurrent exposure to other Aroclor mixtures nearly always occurred, exposure involved dermal as well as inhalation routes (relative

contribution by each route not known), and monitoring data are lacking or inadequate (Alvares et al., 1977; Brown and Jones, 1981; Colombi et al., 1982; Fischbein et al., 1979, 1982, 1985; Fischbein, 1985; Warshaw et al., 1979; Smith et al., 1982; Taylor et al., 1984; Lawton et al., 1985). Insufficient data are available in these studies to determine possible contributions of Aroclor 1254 alone, extent of direct skin exposure and possible contaminants. However, it is relevant to note that dermal and ocular effects, including skin irritation, chloracne, hyperpigmentation and eyelid and conjunctival irritation, have been observed in humans occupationally exposed to Aroclor 1254 and other Aroclor formulations.

Aroclor 1254 was fed to groups of eight female and four male adult rhesus monkeys once daily in dosages of 0, 5, 25 or 100 ug/kg for 14 months, followed by an observation period of 7 months (Levinskas et al., 1984). The Aroclor 1254 was dissolved in corn oil and offered to the animals in apple sauce prior to each day's feeding, and the control mixture (corn oil in applesauce) was used during the observation period. Dosages were adjusted biweekly for changing body weight as necessary. The monkeys were selected on the basis of a successful reproductive history, estimated to be at least 6 years old, and had been in captivity for 2-9 years. After 6 months of treatment the monkeys were bred to untreated males or females from the same colony over an 8-month period and offspring were observed for 2 months. Breeding was continued until conception was diagnosed by digital examination of the uterus and alterations in the menstrual cycle. Evaluations of adult animals included hematology and clinical chemistry. Urinalysis was also performed every 3 months during the study. Semen analyses were performed monthly from just prior to the start of treatment until the end of the treatment period. After 2 months of observation; sperm concentration, total sperm, sperm motility, percent abnormal cells and live/dead ratios were evaluated. Based upon these parameters, no effect was observed upon male reproductive capacity. Necropsies including histological examinations were performed on all adult animals that died during the study or were euthanized at the end of the observation period. Birth weight and somatic measurements were taken for all offspring of exposed females or males. The infants of the exposed females were subsequently evaluated monthly for body weight and complete blood cell counts were performed. Infants that did not show signs of intoxication were euthanized after 2 months and those showing signs were weaned, observed for reversal of signs, and euthanized at the end of the study along with the adults. Necropsies including histological examinations were performed on all infants that died or were euthanized.

Death or euthanasia in extremis occurred in 1/12, 0/12, 1/12 and 5/12 of the adult monkeys in the control, low-, mid- and high-dose groups, respectively. All of the deaths occurred in females except for one male in the high-dose group, and the only deaths considered to be related to treatment were in four of the high-dose animals (3 females, 1 male). Characteristic signs of PCB intoxication developed in the high-dose group after 9 months of exposure, including effects on the eyelids (redness and/or edema, wrinkling) in approximately half the animals and swelling of the lips in all animals. Other characteristic signs included bleeding gums, abnormal fingernail/toenail growth pattern and increased alopecia (including eyelashes) in several of the high-dose monkeys. In general, the signs of intoxication appeared to subside during the post-treatment period. Some of the monkeys in the mid-dose group showed signs of intoxication (swelling of the lips in one male and one female)

after 15 and 18 months, respectively, and alopecia and abnormal nail growth, but no signs attributable to exposure occurred in the low-dose group. Hematologic effects at the high dose were observed including reduced packed cell volume, erythrocyte count, hemoglobin and platelet counts. In addition, increased serum iron and reduced serum cholesterol were observed, particularly in the monkeys that died. Some of the high-dose monkeys also had prolonged bleeding and improper healing at biopsy sites. Dermal histological changes characteristic of PCB poisoning were prominent in the high-dose group, occurring in 11/12 monkeys (8 females, 3 males), and included loss of secretory epithelium in the Meibomian (eyelid) glands and sebaceous glands, partial or total atrophy of sebaceous glands, follicular keratosis and/or squamous cysts. Dermal changes also occurred in four of the mid-dose monkeys, but not in the low-dose or control groups. Other histological alterations included squamous metaplasia in glandular ducts of the tongue or lip (3 high-dose females, 1 high-dose male), subgingival epithelial cysts of the mandible (1 high-dose male, 1 high-dose female, 1 mid-dose male) and hyperplasia in the bile and pancreatic ducts and gall bladder (1 high-dose female). Nonspecific bone marrow alterations (decreased cellularity and/or granulocyte maturation) occurred in 6/12 high-dose monkeys (5 females, 1 male) and may have been compound-related because they correlated with the hematologic changes.

There was no apparent effect on male fertility based on conception rate following matings with the untreated females or the semen analyses (Levinskas et al., 1984). In the female control, low-, mid- and high-dose groups, the numbers of known pregnancies were 7, 7, 7 and 5, respectively, the numbers of live births were 6, 5, 7 and 1, respectively. Analysis of the preceding data showed that there was a statistically significant reduction in fertility in the high-dose group; this analysis refers only to the decreased number of live births. There was a clear exposure-related effect on birth weight and infant body weight gain. When compared with control group infants (mean birth weight 495.2 g) the 0.025 mg/kg-day infants (mean birth weight 392.2 g) showed a statistically significant reduction in birth weight ($p < 0.005$). Most of the infants in the mid-dose group and all of the infants in the high-dose had abnormal clinical signs. These changes included being born with or developed dermal signs that were consistent with those in the adults (e.g., swollen lips, swollen eyelids and/or scanty eyelashes) and more severe at the high dose, and also developed pulmonary signs (e.g., respiratory wheezing). Histological changes in the infants were generally similar to those observed in the adults. These effects included changes in the Meibomian and sebaceous glands, pancreatic ducts and bone marrow. Other histological changes included thymic atrophy in one mid-dose and the high-dose infant, and other effects in the high-dose infant (e.g., retarded kidney cortical maturation, bile duct hyperplasia and gastric mucosal gland cysts).

To summarize the above, no treatment-related effects were observed in the low-dose adults or their infants, indicating that 0.005 mg/kg-day is a NOAEL. For the mid-dose infants there was a 15% reduction in birth weight of infants that was statistically significant ($p < 0.005$). When these infants reached 2 months of age the reduced body weight was 22% below controls and this difference was also found to be statistically significant ($p = 0.05$). Ocular and dermal signs and/or histological changes characteristic of PCB intoxication developed in some adults receiving 25 and 100 ug/kg-day, as well as in most of the infants in these groups. Based on these effects the 0.025 mg/kg-day dosage is a LOAEL. Other effects at the high dose included

decreased adult survival, female fertility and numbers of live births, indicating that 0.1 mg/kg-day is a FEL. This FEL is supported by results of the Truelove study (Truelove et al., 1982).

Aroclor 1254 was fed to 1, 2 or 1 pregnant rhesus monkeys in reported average daily doses of 0, 0.1 or 0.2 mg/kg-day, respectively, 3 days/week for up to 267 days starting on gestation day 60 (Truelove et al., 1982). The exposure period included gestation and lactation. One of the adult monkeys in the low-dose group and the one adult in the high-dose group lost their fingernails after 233 and 242 days of PCB treatment, but other overt signs of intoxication were not observed. There was a significant reduction in antibody production in response to injected SRBC in the exposed monkeys, but levels of antibody production to tetanus toxoid were not appreciably different from control. The two low-dosage monkeys delivered dead infants. The infant of the high-dosage monkey died at age 139 days; this infant showed impaired immune function as assessed by antibody production following SRBC injections. Hematological evaluation performed bimonthly following parturition in adults and the surviving infant were inconclusive. Although evaluation of the dead infants and other results of this study is complicated by the small number of animals, the characteristic dermal sign of PCB toxicity in the exposed monkeys and lack of effects in controls strongly indicate that the developmental toxicity is exposure-related. Therefore, based on the stillbirths, 0.1 mg/kg-day is a FEL in monkeys.

Groups of four young adult female rhesus monkeys were fed 0 or 0.28 mg/kg doses of Aroclor 1254 for 5 days/week for 114-121 weeks (Tryphonas et al., 1986a,b; Arnold et al., 1990). Groups of four mature adult female cynomolgus monkeys that had a poor breeding history were similarly exposed for 55-58 weeks (Tryphonas et al., 1986a; Arnold et al., 1990). The Aroclor mixture contained no detectable polychlorinated dibenzo-p-dioxin contaminants. Adjusting for the partial weekly exposure gives an average daily dosage of 0.2 mg/kg-day. Prominent clinical signs appeared in all exposed rhesus monkeys during the first 2-12 months of exposure, including facial and periorbital edema, loss of eyelashes, Meibomian gland enlargement and impaction, conjunctivitis, nail lesions progressing from dryness to detachment and gingival hyperplasia and necrosis of varying severity. Two of the exposed rhesus monkeys developed overwhelming infections of the eye or periodontal tissue after 27 months of exposure prompting sacrifice within 48 hours. The hematology and serum biochemistry evaluations showed various changes in the exposed rhesus monkeys, particularly slight or moderate normocytic anemia, depressed erythropoiesis in bone marrow and increased triglycerides and SGOT. The immunologic testing was inconclusive due to large interspecies variability. Pathology findings in the exposed rhesus monkeys included effects in the liver of three monkeys (30-55% increased relative liver weight, hepatocellular hypertrophy and necrosis, bile duct epithelial hypertrophy and hyperplasia, gall bladder epithelial hypertrophy), thyroid of two monkeys (enlargement, occasional follicular cell desquamation) and stomach of two monkeys (hypertrophic gastropathy). The cynomolgus monkeys had effects that were generally consistent with but less extensive and severe than those observed in the rhesus monkeys. After 38 weeks of exposure the rhesus monkeys were mated with untreated males; cynomolgus monkeys were not mated. The control and exposed rhesus monkeys became pregnant within 7 and 8 matings, respectively. Following extended post-implant bleeding all of the treated rhesus monkeys aborted within 30-60 days of gestation. Following recovery

from the abortions the monkeys were bred again up to a maximum of seven times but none appeared to conceive. The menstrual cycle lengths and durations became erratic and longer during and subsequent to the breeding. Based on the abortions, reproductive impairment and pronounced overt signs of toxicity, the 0.2 mg/kg-day dosage is an FEL in monkeys.

Aulerich and Ringer (1977) performed a breeding study in which groups of eight female and two male adult mink were fed diets containing 0 or 2 ppm Aroclor 1254 for 39 weeks or until the kits were 4 weeks of age. The Aroclor was dissolved in acetone which was evaporated from the diet prior to feeding. Using assumed values of 150 g/day for food consumption and 0.8 kg for body weight for female mink (Bleavins et al., 1980), the estimated dosage of Aroclor 1254 is 0.4 mg/kg-day. Approximately monthly determinations reportedly showed no statistically significant ($p < 0.05$) differences between the control and treated mink in body weight gain, hemoglobin, and hematocrit. Only two of seven mated females gave birth, producing one infant each. Of the two infants, one was born dead and the other had low body weight and was dead by age 4 weeks. Based on the reproductive and/or fetal toxicity resulting in nearly complete lack of births, 0.4 mg/kg-day is a FEL for Aroclor 1254 in mink.

Twelve female and four male adult ranch-bred mink (age 8 months, body weight not reported) were fed a diet containing 1 ppm Aroclor 1254 for 6 months (Wren et al., 1987a,b). Groups of 15 females and five males were used for unexposed controls. The mink were bred after approximately 12-14 weeks of exposure and exposure was continued until weaning at age 5 weeks. Using assumed values for food consumption and for body weight for female mink (Bleavins et al., 1980), the estimated dosage of Aroclor 1254 is 0.15 mg/kg-day. Offspring mortality during the first week of life was 75.8% higher in the exposed group than in the controls. Average body weight was significantly lower in the exposed offspring at age 3 and 5 weeks, but not at age 1 week, suggesting that transfer of PCBs by lactation may have contributed to the effect. There were no exposure-related effects on adult survival or mating performance, number of offspring per female mated or female that delivered, adult thyroid plasma T3 or T4 levels during the exposure period, adult scrotal diameter, offspring survival or relative liver weight at weaning or organ weights or histology (brain, kidney, adrenal, pituitary, thyroid). Teratogenicity was not evaluated. The neonatal mortality indicates that 0.15 mg/kg-day is an FEL in mink.

Groups of 10 female Sprague-Dawley rats were fed 0, 1, 5, 10 or 50 ppm Aroclor 1254 in the diet for approximately 5-6 months (Byrne et al., 1987). The Aroclor was dissolved in acetone which was evaporated from the diet prior to feeding. Based on reported body weight and food consumption data the dosages are estimated to be 0.09, 0.43, 0.61 and 4.3 mg/kg-day. Serum thyroxine (T4) and triiodothyronine (T3) were evaluated at five different times during 140 and 175 days of treatment, respectively. Serum T4 levels were significantly reduced at 0.09 and 0.43 mg/kg-day by day 35 and at greater than or equal to 0.61 mg/kg-day by day 14. T3 levels were significantly reduced at 0.09 mg/kg-day by day 40 and at greater than or equal to 0.4 mg/kg-day by day 20. The suppressions were generally dose-related for T4 throughout the treatment period and T3 after 75 days. Disappearance rate of injected L-[125I] T4 was significantly decreased at greater than or equal to 0.09 mg/kg-day. Rats treated with only 0.43 or 0.61 mg/kg-day for approximately 5 months

and challenged with i.p. injected TSH had diminished response of serum T4 and T3. Thyroid histology was not evaluated. There were no treatment-related effects on relative thyroid weight, body weight or food consumption. The findings of this study indicate that the decreased serum T3 and T4 resulted primarily from direct damage to the thyroid rather than suppression of the hypothalamo-pituitary axis or any enhanced peripheral catabolism (e.g., liver). Insufficient data are available to determine if the decreases in circulating thyroid hormones were physiologically significant. However, because the effects are indicative of impaired organ function, they are at least potentially adverse and 0.09 mg/kg-day is considered to represent a LOAEL in rats.

Groups of 10 female Sprague-Dawley rats were fed 0, 1, 5, 10 or 50 ppm Aroclor 1254 in the diet for 5 months (Byrne et al., 1988). The Aroclor was dissolved in acetone which was evaporated from the diet prior to feeding. Using a rat food consumption factor of 0.05 kg food/kg body weight, the dosages are estimated to be 0.05, 0.25, 0.5 and 2.5 mg/kg-day. Serum levels of adrenal cortex hormones were evaluated in 8-10 rats 3-5 times during the treatment period. Serum corticosterone was significantly ($p < 0.05$) decreased at greater than or equal to 0.25 mg/kg-day after approximately 60 days of exposure. Serum dehydroepiandrosterone and dehydroepiandrosterone sulfate were significantly ($p < 0.05$) decreased at 0.25 and 0.5 mg/kg-day (not evaluated at other dosages) after approximately 100 days and 25 days of exposure, respectively. Serum corticosterone is the principal glucocorticoid in rats. Adrenal weight, adrenal histology and non-adrenal endpoints other than food consumption were not evaluated. Food consumption did not significantly differ between and among control and treatment groups. The results of this study are suggestive of toxicity to the adrenal rather than response to stress which would be expected to increase the release of glucocorticoids. Insufficient data are available to determine if the decreases in circulating adrenal cortex hormones were physiologically significant. However, because the effects are indicative of impaired organ function, they are at least potentially adverse. The dosages of 0.05 and 0.25 mg/kg-day therefore are considered to represent a NOEL and LOAEL, respectively, in rats.

Hepatotoxicity is a prominent effect of Aroclor 1254 that is well characterized in rats (U.S. EPA, 1990). The spectrum of effects includes hepatic microsomal enzyme induction, increased serum levels of liver-associated enzymes indicative of possible hepatocellular damage, liver enlargement, lipid deposition, fibrosis and necrosis. Estimated subchronic dosages as low as 1.25-2.5 mg/kg-day have been reported to produce increased liver weight and hepatic biochemical alterations in rats, but the lowest dosages producing signs of hepatic effects are generally higher than the lowest dosages that caused thyroid, adrenal and bone changes (Litterset et al., 1972; Bruckner et al., 1974; Kling and Gamble, 1982; Andrews et al., 1989). Rats fed 6.8 mg/kg-day for 8 months (Kimbrough et al., 1972) or an estimated dosage of 50 mg/kg-day for 30 days (Kling et al., 1978) developed fatty and necrotic degenerative hepatic histologic changes. Chronic dietary exposure to 1.25-5 mg/kg-day for approximately 2 years produced only preneoplastic and neoplastic liver lesions in rats (NCI, 1978; Ward, 1985).

A two-generation reproduction study was performed in which groups of 20 female and 10 male Sherman rats (age 3-4 weeks, body weight not reported) were fed 0, 1, 5, 20 or 100 ppm dietary Aroclor 1254 (Monsanto Lot No. AK-38) in

peanut oil vehicle (Linder et al., 1974). Reported dosages were 0.06, 0.32, 1.5 and 7.6 mg/kg-day, and different controls were used for the less than or equal to 0.32 and greater than or equal to 1.5 mg/kg-day groups. Exposure times (before mating or conception-to-mating) ranged from 62-274 days. Exposure-related effects included increased relative liver weight in F1a weanlings at greater than or equal to 0.06 mg/kg-day, enlarged and vacuolated hepatocytes in F2a weanlings at greater than or equal to 1.5 mg/kg-day, and 15-72% reduced litter size at greater than or equal to 1.5 mg/kg-day in the F1b, F2a and F2b generations and at 7.6 mg/kg-day in the F1a generation. Relative testes weights were increased in adult F1b males at 7.6 mg/kg-day (other groups not evaluated). The highest NOAEL is 0.32 mg/kg-day based on the increased liver weight without altered histology. The decreased litter size indicates that 1.5 mg/kg-day is a FEL.

A one-generation reproduction study was performed in which groups of 10 male and 10 female Sherman rats were fed 0, 100 or 500 ppm dietary Aroclor 1254 for 67 or 186 days prior to pair-mating for the F1a and F1b generations, respectively (Linder et al., 1974). The F0 rats received reported dosages of 0, 7.2 and 37.0 mg/kg-day and were sacrificed after a total exposure duration of 8 months for hematology, organ weight and liver histology evaluation. The study was terminated after the F1b pups were weaned. Effects in the P1 rats included increased liver weight in both sexes greater than or equal to 7.2 mg/kg-day, increased relative testis weight (absolute weight unchanged) at 37.0 mg/kg-day, decreased body weight gain in both sexes at 37.0 mg/kg-day, and changes in hematological values (reduced hematocrit and hemoglobin in both sexes, increased total leukocytes with normal differential count in females) at 37.0 mg/kg-day. Specific information on liver pathology was not reported but degenerative changes similar to those found in the Kimbrough et al. (1972) subchronic study were indicated for both dosages. Effects on the offspring included reduced survival to weaning at 7.2 mg/kg-day (85.9 and 68.1% survival in F1a and F1b pups, respectively, compared with 95.5% in controls), and reduced litter size and number and 100% pup mortality by day 3 in F1a rats at 37.0 mg/kg-day. The decreases in postnatal survival indicate that both dosages are FELs.

Groups of six to eleven female Wistar rats were fed 2.5, 26 or 269 ppm Aroclor 1254 in the diet during gestation and lactation (Overman et al., 1987). A control group was fed untreated diet that contained 0.02 ppm PCBs (i.e., no added PCBs). Using a rat food consumption factor of 0.05 kg food/kg body weight, the dosages are estimated to be 0.001, 0.13, 1.3 and 13.5 mg/kg-day. The following neurobehavioral endpoints were significantly delayed or reduced in the pups: appearance of the auditory startle response at 0.13 and 1.3 mg/kg-day at age 6 days (slightly delayed), development of righting ability at 1.3 mg/kg-day at days of age (slightly delayed) and performance on a motor coordination test at 1.3 mg/kg-day at age 7 and 8 days (slower performance). Grip strength and appearance of eye opening were not affected by exposure. Other effects attributable to exposure included increased relative liver weight in pups at weaning at greater than or equal to 1.3 mg/kg-day and reduced birth weight, 50% mortality by 2 days of age and retarded growth in pups at 13.5 mg/kg-day. There were no exposure-related effects on maternal weight gain, gestation length, litter size, pup sex ratios, number of live and dead pups or physical appearance, relative spleen and thymus weight or relative and absolute brain weight of pups. Brain PCB levels increased from birth to weaning in all groups. Based on the evidence

for impaired motor coordination in developing infants the 0.13 and 1.3 mg/kg-day dosages are a NOAEL and LOAEL, respectively.

Dietary Aroclor 1254 was administered to groups of 4-10 female ICR mice in concentrations of 0, 1, 10 or 100 ppm from 90 days before mating through gestation day 18 (Welsch, 1985). The investigators estimated the dosages to be 0.125, 1.25 and 12.5 mg/kg-day. No developmental toxicity was observed as judged by number of litters, number of dead and reabsorbed fetuses, fetal weight, incidence of gross malformations or skeletal development. Fetuses were not examined for internal malformations. Maternal effects other than significantly increased relative liver weight at greater than or equal to 0.125 mg/kg-day were not observed. No developmental effects were observed in mice treated with the same doses of PCB only on gestation days 6-18. Based on the increased maternal liver weight the highest NOAEL is 12.5 mg/kg-day.

Groups of seven adult male New Zealand white rabbits were fed dietary Aroclor 1254 in reported estimated dosages of 0, 0.18, 0.92, 2.10 or 6.54 mg/kg-day for 8 weeks (Street and Sharma, 1975). Immunological testing was started after 4 weeks of treatment at which time the rabbits were immunized with injected SRBCs. No treatment-related changes in serum antibody titers to SRBC (hemolysin and hemagglutination) were observed. SRBC-induced increases in serum gamma-globulin were consistently but not statistically significantly decreased by exposure, and the number of globulin-producing cells in popliteal lymph nodes was significantly decreased at 0.92 and 6.54 mg/kg-day. Skin sensitivity to tuberculin was generally lower in the treated groups but none of the decreases were statistically significant. Marked histologic atrophy of the thymus cortex was observed at 0.18 mg/kg-day and higher dosages except 0.92 mg/kg-day. There were no treatment-related effects on leukocyte count, histology of the spleen, thymus, liver, kidneys or spleen, relative kidney or adrenal weight, terminal body weight or food consumption. Relative liver and spleen weights were significantly increased at greater than or equal to 2.10 mg/kg-day; the increase in liver weight was 74% at the highest dosage. The 0.18 mg/kg-day dosage is a LOAEL based on the thymic cortical atrophy.

Limited specific information is available on the oral absorption of Aroclor 1254. Pregnant ferrets that ingested a single oral dose of Aroclor 1254 (approximately 0.06 mg/kg) absorbed approximately 85% of the initial amount (Bleavins et al., 1984). Studies predominately of individual chlorobiphenyl congeners indicate, in general, that PCBs are readily and extensively absorbed by animals. These studies have found oral absorption efficiency on the order of 75 to >90% in rats, mice and monkeys (Albro and Fishbein, 1972; Allen et al., 1974; Tanabe et al., 1981; Clevenger et al., 1989). A study of a non-Aroclor 54% chlorine PCB mixture prepared by the investigators provides direct evidence of absorption of PCBs in humans after oral exposure (Buhler et al., 1988), and indirect evidence of oral absorption of PCBs by humans is available from studies of ingestion of contaminated fish by the general population (Schwartz et al., 1983; Kuwabara et al., 1979). There are no quantitative data regarding inhalation absorption of PCBs in humans but studies of workers exposed suggest that PCBs are well absorbed by the inhalation and dermal routes (Maroni et al., 1981a,b; Smith et al., 1982; Wolff, 1985). PCBs distribute preferentially to adipose tissue and concentrate in human breast milk due to its high fat content (Jacobson et al., 1984b; Ando et al., 1985).

The metabolism of PCBs following oral and parenteral administration in animals has been extensively studied and reviewed, but studies in animals following inhalation or dermal exposure are lacking (Sundstrom and Hutzinger, 1976; Safe, 1980; Sipes and Schnellmann, 1987). Information on metabolism of PCBs in humans is limited to occupationally exposed individuals whose intake is derived mainly from inhalation and dermal exposure (Jensen and Sundstrom, 1974; Wolff et al., 1982; Schnellmann et al., 1983; Safe et al., 1985; Fait et al., 1989). In general, metabolism of PCBs depends on the number and position of the chlorine atoms on the phenyl ring of the constituent congeners (i.e., congener profile of the PCB mixture) and animal species. Although only limited data are available on metabolism of PCBs following inhalation exposure, there is no reason to suspect that PCBs are metabolized differently by this route.

Data exist on the in vitro hepatic metabolism and in vivo metabolic clearance of 2,2',3,3',6,6'-hexachlorobiphenyl and 4,4'-dichlorobiphenyl congeners in humans, monkeys, dogs and rats (Schnellmann et al., 1985). The hexachlorobiphenyl congener is a constituent of Aroclor 1254. For each congener, the Vmax values for metabolism in the monkey, dog and rat are consistent with the respective metabolic clearance values found in vivo. Thus, the kinetic constants for PCB metabolism obtained from the dog, monkey and rat hepatic microsomal preparations were good predictors of in vivo metabolism and clearance for these congeners. In investigations directed at determining which species most accurately predicts the metabolism and disposition of PCBs in humans, the in vitro metabolism of these congeners was also studied using human liver microsomes (Schnellmann et al., 1983, 1984). Available data suggest that metabolism of PCBs in humans would most closely resemble that of the monkey and rat. For example, the in vitro apparent Km and Vmax are comparable between humans and monkeys. These studies show consistency between the in vitro and in vivo findings and collectively indicate that metabolism of the two congeners is similar in monkeys and humans.

o ORAL RFD CONFIDENCE :

Study – Medium
Data Base – Medium
RfD – Medium

Confidence in the principal study is medium. Groups of 16 rhesus monkeys were tested at four dose levels and LOAEL was established on the basis of clinical signs and immunologic alterations. Data for female and male reproductive function and developmental data in a nonhuman primate species is taken from an unpublished study (Levinskas et al., 1984) which established a NOAEL for reproductive effects at 0.005 mg/kg-day. The Arnold study also included evaluation of reproductive function but the data have not been completely analyzed. Preliminary examination of the Arnold et al. data indicate that the LOAEL for female reproductive function may be 0.005 mg/kg-day. This inconsistency in effect levels for reproductive toxicity was viewed as a limitation to the data base. Furthermore, there is a limitation in the characterization of reproductive toxicology because results of an unpublished study have been considered. An extensive number of laboratory animal and human studies were available for review, including two-generation reproductive studies. The chronic, 2-year bioassays performed in F344 rats showed evidence

of degenerative hepatocellular changes in addition to the neoplastic changes that were observed. Only limited assessment of nonhepatic changes were made. Human occupational and environmental data is available for commercial PCB mixtures in general but not specifically for Aroclor 1254. The data base is rated medium on the basis of these considerations. Overall confidence in the RfD is medium.

o ORAL RFD SOURCE DOCUMENT :

Source Document – This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation – U.S. EPA, 1984, 1989, 1990

o REVIEW DATES : 06/16/93, 02/16/94
o VERIFICATION DATE : 02/16/94
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RDI - NO DATA
CAREV- NO DATA
CARO - NO DATA
CARI - NO DATA
CARDR- NO DATA

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU- NO DATA

WQCAQ- NO DATA

MCLG - NO DATA

MCL - NO DATA

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC - NO DATA

SARA - NO DATA

RCRA - NO DATA

TSCA - NO DATA

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- IREF - None
- CREF - None
- HAREF - None

PCB 1248

1 - IRIS
NAME - Aroclor 1248
RN - 12672-29-6
IRSN - 631
DATE - 940406
UPDT - 04/06/94, 5 fields
STAT - Oral RfD Assessment (RDO) message 04/01/94
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
IRH - 09/01/92 RDO Oral RfD now under review
IRH - 12/01/92 RDO Work group review date added
IRH - 07/01/93 RDO Work group review date added
IRH - 08/01/93 RDO Work group review date added
IRH - 04/01/94 RDO Oral RfD message on-line
IRH - 04/01/94 OREF Oral RfD references on-line
RLEN - 10476SY - Aroclor 1248
SY - HSDB 6356

RDO -
o ORAL RFD SUMMARY :

The health effects data for Aroclor 1248 were reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for the derivation of an oral RfD. The verification status for this chemical currently is NOT VERIFIABLE. For additional information on the health effects of this chemical, interested parties are referred to the U.S. EPA documentation listed below.

NOT VERIFIABLE status indicates that the U.S. EPA RfD/RfC Work Group deemed the data base at the time of review to be insufficient to derive an oral RfD according to the current Agency guidelines. This status does not preclude the use of information in cited references for assessment by others.

Derivation of an oral RfD for Aroclor 1248 is not recommended because a Frank Effect (death of an infant) was noted at the lowest dose tested in a sensitive animal species, rhesus monkeys (*Macaca mulatta*). In general, Rhesus monkeys have shown adverse effects to PCB mixtures at doses 10-fold lower than in other species. The data indicated a dose-response relationship for this effect.

Schantz et al. (1989) evaluated neurobehavioral performance in offspring of rhesus monkeys that had been exposed to 0.03, 0.1 and 0.2 mg/kg-day of dietary Aroclor 1248 for different durations. Group I consisted of infants whose dams had received 0.03 mg/kg-day. Of the seven dams for this group, six delivered viable offspring. Necropsy of the infant who died at the time of weaning showed signs of PCB intoxication that included thymic atrophy and skin hyperpigmentation. Group II consisted of offspring of 4/8 females fed 0.1 mg/kg-day of Aroclor 1248. Of the eight dams of this group, one delivered a dead infant and one delivered an infant that died shortly after weaning with signs of PCB intoxication. Group III consisted of offspring of 3/7 females fed 0.2 mg/kg-day of Aroclor 1248. Of the seven females that were dams in this group, only three delivered live infants. Information on maternal

toxicity was not provided in the report. Mild dermatological lesions and hyperpigmentation about the hairline developed in offspring in all treated groups during nursing, but no signs of toxicity were evident at the time of neurological testing (age 14 months). Offspring weights at birth and weaning were significantly reduced in Group III. Offspring in Groups I and II did not differ from controls on spatial, color or shape in two-choice discrimination reversal learning tests, but decreased performance on a shape discrimination problem was observed in Group III when irrelevant cues were inserted. On the basis of thymic atrophy and chloracne and death of 1 of 7 infants, it is concluded that 0.03 mg/kg-day represents a FEL for developmental effects.

Adult female Rhesus monkeys were fed 0, 2.5 or 5 ppm (0, 0.1 and 0.2 mg/kg-day) of Aroclor 1248 incorporated in food pellets for up to 14 months (Barsotti et al., 1976; Barsotti, 1980). The exposure period ran from 7 months prior to breeding through gestation, and then for an additional 4 months until the infants were weaned. Some treated females began showing skin changes, such as hyperpigmentation and alopecia, characteristic signs of PCB intoxication, during the first 2 months of dosing. Monkeys with less body fat were the first to show clinical signs, regardless of the dose group to which they were assigned. All treated females showed signs of PCB intoxication to some degree by 6 months. A progressive increase in SGPT values was observed for all treated monkeys and this increase was found to be statistically significant ($p < 0.05$) by the 22nd month of the study, even though dosing stopped at the end of the 14th month. One female in each dose group developed severe shigellosis and died, and other dosed females developed clinical signs of shigellosis but did not die. Necropsies of deceased monkeys showed focal necrosis and lipid deposition of the liver, as well as marked subcutaneous edema. Increased menstrual duration was noted as well as occasional amenorrhea.

For the experimental breeding trial, conducted during the dosing period, all low-dose monkeys (8/8) conceived; 3/8 aborted and 5/8 delivered live infants. However, 3 of these 5 liveborn infants showed clinical signs of PCB toxicity and, being unable to withstand the stress of weaning, died when separated from their dams. Among the high-dose monkeys, 6/8 conceived. Among these six conceptions, four ended in abortion, one infant went to term, but was stillborn. Only one normal birth occurred among this group; however, at the time of weaning, this infant showed clinical signs of PCB toxicity and died.

The investigators realized that PCB mixtures might have latent effects that could appear long after dosing had ceased. Thus, they included three additional recovery breeding periods after dosing had been completed.

The first recovery breeding trial occurred approximately 22 months after the initiation of Aroclor 1248 dosing and 8 months after dosing had stopped. For the low-dose dams, 8/8 conceived. One of these eight conceptions resulted in abortion. Of the seven livebirths, two infants died at or before weaning. Among the high-dose mothers, 7/7 conceived. There was one abortion and one stillbirth among this group of seven mothers, and five livebirths. Among the group of five livebirths, three infants died at or before weaning.

A second recovery breeding trial was conducted approximately 36 months after the completion of Aroclor 1248 dosing. Among the low-dose mothers, 5/7

conceived. There was one stillbirth and four live births. All four of the liveborn infants survived past weaning and were available for behavioral testing at 14 months and 4 years of age. Among the high-dose mothers, 4/6 conceived for this breeding trial. There were no abortions among the four conceptions, but one stillbirth did occur; there were three livebirths.

The third recovery breeding trial was conducted 55 months after the completion of Aroclor 1248 dosing. Among the low-dose dams, 7/7 conceived. There were no abortions among this group but two stillbirths did occur. All five liveborn infants survived past weaning. For the high-dose mothers, only five had normal reproductive cycles and 4/5 conceived. Among the four conceptions, one ended in abortion, another infant was stillborn and two were born live.

In the first recovery breeding trial the average birth weights for the dosed groups were found to be reduced when compared with controls. For the second recovery breeding trial, the mean weight of the test group infants was 15 and 22% below the control group.

Results of this prolonged recovery period revealed impairment of reproductive function in female Rhesus monkeys lasting for more than 4 years after dosing ceased. In the groups of infants for which birth-weight data are available, a significant reduction in mean birth weight for PCB-exposed infants is evident.

Thomas and Hinsdill (1978) performed immunologic tests after Rhesus monkeys had been fed 0, 2.5 and 5 ppm dietary Aroclor 1248 for 11 months. All treated monkeys developed facial acne and edema and swollen eyelids to varying degrees after 6 months, with pronounced alopecia occurring in the 0.2 mg/kg-day group. Following the treatment period, the monkeys were inoculated with sheep red blood cells (SRBC) and tetanus toxoid. Anti-SRBC antibody titers were significantly reduced in the 0.2 mg/kg-day group at weeks 1 and 12 after inoculation, but antibody response to tetanus toxoid was not affected by treatment at either dosage level.

Groups of three female New Zealand white rabbits were fed 0, 10, 100 or 250 ppm of Aroclor 1248 for 4 weeks and bred with untreated males (Thomas and Hinsdill, 1980). No maternal toxicity was evident. Body-weight gain was significantly reduced in the offspring in the high-dose group.

Barsotti, D.A., R.J. Marlar and J.R. Allen. 1976. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). *Food Cosmet. Toxicol.* 14: 99-103.

Barsotti, D.A. 1980. *Gross, Clinical, and Reproductive Effects of Polychlorinated Biphenyls in the Rhesus Monkey*. August. Ph.D. Thesis, available through the University Library, University of Wisconsin, Madison, WI.

Schantz, S.L., E.D. Levin, R.W. Bowman et al. 1989. Effects of perinatal PCB exposure on discrimination-reversal learning in monkeys. *Neurotoxicol. Teratol.* 11: 243-250.

Thomas, P.T. and R.D. Hinsdill. 1978. Effect of polychlorinated biphenyls on

the immune responses of rhesus monkeys and mice. Toxicol. Appl. Pharmacol. 44: 41-51.

Thomas, P.T. and R.D. Hinsdill. 1980. Perinatal PCB exposure and its effect on the immune system of young rabbits. Drug Chem. Toxicol. 3: 173-184.

o REVIEW DATES : 08/12/92, 11/04/92, 06/16/93, 07/20/93

RDI - NO DATA
CAREV- NO DATA
CARO - NO DATA
CARI - NO DATA
CARDR- NO DATA

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU- NO DATA

WQCAQ- NO DATA

MCLG - NO DATA

MCL - NO DATA

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC - NO DATA

SARA - NO DATA

RCRA - NO DATA

TSCA - NO DATA

OREF - Barsotti, D.A., R.J. Marlar and J.R. Allen. 1976. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). Food Cosmet. Toxicol. 14: 99-103.

OREF - Barsotti, D.A. 1980. Gross, Clinical, and Reproductive Effects of Polychlorinated Biphenyls in the Rhesus Monkey. August. Ph.D. Thesis, available through the University Library, University of Wisconsin, Madison, WI.

OREF - Schantz, S.L., E.D. Levin, R.W. Bowman et al. 1989. Effects of perinatal PCB exposure on discrimination-reversal learning in monkeys. Neurotoxicol. Teratol. 11: 243-250.

OREF - Thomas, P.T. and R.D. Hinsdill. 1978. Effect of polychlorinated biphenyls on the immune responses of rhesus monkeys and mice. Toxicol. Appl. Pharmacol. 44: 41-51.

OREF - Thomas, P.T. and R.D. Hinsdill. 1980. Perinatal PCB exposure and its effect on the immune system of young rabbits. Drug Chem. Toxicol. 3: 173-184.

IREF - None

CREF - None

HAREF - None

PCB 1260

NOTE: PCB 1260 is not listed on IRIS. This printout pertains to the toxicities of PCBs in general.

1 - IRIS
NAME - Polychlorinated biphenyls (PCBs)
RN - 1336-36-3
IRSN - 288
DATE - 940601
UPDT - 06/01/94, 3 fields
STAT - Oral RfD Assessment (RDO) message 06/01/94
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 01/01/90
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 05/01/89 CAR Carcinogen summary on-line
IRH - 01/01/90 CAR Text edited
IRH - 01/01/90 REFS Bibliography on-line
IRH - 01/01/92 EXSR Regulatory Action section on-line
IRH - 06/01/94 RDO Message only
RLEN - 24527
SY - AROCLOR
SY - AROCLOR 1221
SY - AROCLOR 1232
SY - AROCLOR 1242
SY - AROCLOR 1248
SY - AROCLOR 1254
SY - AROCLOR 1260
SY - AROCLOR 1262
SY - AROCLOR 1268
SY - AROCLOR 2565
SY - AROCLOR 4465
SY - AROCLOR 5442
SY - BIPHENYL, POLYCHLORO-
SY - CHLOPHEN
SY - CHLOREXTOL
SY - CHLORINATED BIPHENYL
SY - CHLORINATED DIPHENYL
SY - CHLORINATED DIPHENYLENE
SY - CHLORO BIPHENYL
SY - CHLORO 1,1-BIPHENYL
SY - CLOPHEN
SY - DYKANOL
SY - FENCLOL
SY - INERTEEN
SY - KANECHLOR
SY - KANECHLOR 300
SY - KANECHLOR 400
SY - MONTAR
SY - NOFLAMOL
SY - PCB
SY - PCBs
SY - PHENOCHLOR
SY - PHENOCLOL
SY - POLYCHLORINATED BIPHENYL
SY - Polychlorinated Biphenyls
SY - POLYCHLOROBIPHENYL
SY - PYRALENE

SY - PYRANOL
SY - SANTOTHERM
SY - SANTOTHERM FR
SY - SOVOL
SY - THERMINOL FR-1
SY - UN 2315

RDO -

o ORAL RFD SUMMARY :

Please check the following individual aroclor files for RfD assessments:
Aroclor 1016, Aroclor 1248, Aroclor 1254, Aroclor 1260.

RDI - NO DATA

CAREV-

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : hepatocellular carcinomas in three strains of rats and two strains of mice and inadequate yet suggestive evidence of excess risk of liver cancer in humans by ingestion and inhalation or dermal contact.
- o HUMAN CARCINOGENICITY DATA :

Inadequate. Although there are many studies, the data are inadequate due to confounding exposures or lack of exposure quantification. The first documentation of carcinogenicity associated with PCB exposure was reported at a New Jersey petrochemical plant involving 31 research and development employees and 41 refinery workers (Bahn et al., 1976, 1977). Although a statistically significant increase in malignant melanomas was reported, the two studies failed to report a quantified exposure level and to account for the presence of other potential or known carcinogens. In an expanded report of these studies, NIOSH (1977) concurred with the Bahn et al. (1976) findings.

Brown and Jones (1981) reported a retrospective cohort mortality study on 2567 workers who had completed at least 3 months of employment at one or two capacitor manufacturing plants. Exposure levels were 24-393 mg/cu.m at plant A and 318-1260 mg/cu.m at plant B. No excess risk of cancer was observed. In a 7-year follow-up study, Brown (1987) reported a statistically significant excess risk of liver and biliary cancer, with four of the five liver cancers in female workers at plant B. A review of the pathology reports indicated that two of the liver tumors counted in the follow-up study were not primary liver tumors. When these tumors are excluded the elevation in incidence is not statistically significant. The results also may be confounded by population differences in alcohol consumption, dietary habits, and ethnic composition.

Bertazzi et al. (1987) conducted a mortality study of 544 male and 1556 female employees of a capacitor-making facility in Northern Italy. Aroclor 1254 and Pyralene 1476 were used in this plant until 1964. These were progressively replaced by Pyralenes 3010 and 3011 until 1970, after which

lower chlorinated Pyralenes were used exclusively. In 1980 the use of PCBs was abandoned. Some employees also used trichloroethylene but, according to the authors, were presumed to be protected by efficient ventilation. Air samples were collected and analyzed for PCBs in 1954 and 1977 because of reports of chloracne in workers. Quantities of PCBs on workers' hands and workplace surfaces also were measured in 1977. In 18 samples, levels ranged from 0.2-159.0 ug/sq.m on workplace surfaces and 0.3-9.2 ug/sq.m on workers' hands.

The authors compared observed mortality with that expected between 1946 and 1982 based on national and local Italian mortality rates. With vital status ascertainment 99.5% complete, relatively few deaths were reported by 1982 [30 males (5.5%) and 34 females (2.2%)]. In cohort males, the number of deaths from malignant tumors was significantly higher than expected compared with local or national rates, as was the number of deaths from cancer of the GI tract (6 observed vs. 1.7 national expected and 2.2 local expected). Of the six GI cancer deaths, one was due to liver cancer and one to biliary tract cancer. Deaths from hematologic neoplasms in males were also higher than expected, but the excess was not statistically significant. Total cancer deaths in females were significantly elevated in comparison to local rates (12 observed vs. 5.3 expected). None of these were liver or biliary cancers. The number of deaths from hematologic neoplasms in females was higher than expected when compared with local rates (4 observed vs. 1.1 expected). This study is limited by several factors, particularly the small number of deaths that occurred by the cut-off period. The power of the study is insufficient to detect an elevated risk of site-specific cancer. In addition, the authors stated, after an examination of the individual cases, that interpretation of the increase in GI tract cancer in males was limited, as it appeared likely that some of these individuals had only limited PCB exposure. Confounding factors may have included possible contamination of the PCBs by dibenzofurans and exposure of some of the workers to trichloroethylene, alkylbenzene, and epoxy resins.

Two occurrences of ingestion of PCB-contaminated rice oil have been reported: the Yusho incident of 1968 in Japan and the Yu-Cheng incident of 1979 in Taiwan. Amano et al. (1984) completed a 16-year retrospective cohort mortality study of 581 male and 505 female victims of the Yusho incident. A consistently high risk of liver cancer in females over the entire 16 years was observed; liver cancer in males was also significantly increased. Several serious limitations are evident in this study. There was a lack of information regarding job histories or the influence of alcoholism or smoking.

The information concerning the diagnosis of liver cancer was obtained from the victims' families, and it is not clear whether this information was independently verified by health professionals. For some of the cancers described, the latency period is shorter than would be expected. Furthermore, the contaminated oils contained polychlorinated dibenzofurans and polychlorinated quinones as well as PCBs, and the study lacks data regarding exposure to the first two classes of compounds. There is strong evidence indicating that the health effects seen in Yusho victims were due to ingestion of polychlorinated dibenzofurans, rather than to PCBs themselves (reviewed in EPA, 1988). The results of the Amano et al. study can, therefore, be considered as no more than suggestive of carcinogenicity of PCBs.

o ANIMAL CARCINOGENICITY DATA :

Sufficient. PCB mixtures assayed in the following studies were commercial preparations and may not be the same as mixtures of isomers found in the environment. Although animal feeding studies demonstrate the carcinogenicity of commercial PCB preparations, it is not known which of the PCB congeners in such preparations are responsible for these effects, or if decomposition products, contaminants or metabolites are involved in the toxic response. Early bioassays with rats (Kimura and Baba, 1973; Ito et al., 1974) were inadequate to assess carcinogenicity due to the small number of animals and short duration of exposure to PCB. A long-term bioassay of Aroclor 1260 reported by Kimbrough et al. (1975) produced hepatocellular carcinomas in female Sherman rats when 100 ppm was administered for 630 days to 200 animals.

Hepatocellular carcinomas and neoplastic nodules were observed in 14 and 78%, respectively, of the dosed animals, compared with 0.58 and 0%, respectively, of the controls.

The NCI (1978) reported results for 24 male and 24 female Fischer 344 rats treated with Aroclor 1254 at 25, 50, or 100 ppm for 104 to 105 weeks. Although carcinomas of the gastrointestinal tract were observed among the treated animals only, the incidence was not statistically significantly elevated. An apparent dose-related incidence of hepatic nodular hyperplasia in both sexes as well as hepatocellular carcinomas among mid- to high-dose treated males was reported (4-12%, compared to 0% in controls).

Norback and Weltman (1985) fed 70 male and 70 female Sprague-Dawley rats a diet containing Aroclor 1260 in corn oil at 100 ppm for 16 months, followed by a 50 ppm diet for an additional 8 months, then a basal diet for 5 months. Control animals (63 rats/sex) received a diet containing corn oil for 18 months, then a basal diet alone for 5 months. Among animals that survived for at least 18 months, females exhibited a 91% incidence (43/47) of hepatocellular carcinoma. An additional 4% (2/47) had neoplastic nodules. In males corresponding incidences were 4% (2/46) for carcinoma and 11% (5/46) for neoplastic nodules. Concurrent liver morphology studies were carried out on tissue samples obtained by partial hepatectomies of three animals/group at eight time points. These studies showed the sequential progression of liver lesions to hepatocellular carcinomas.

Orally administered PCB resulted in increased incidences of hepatocellular carcinomas in two mouse strains. Ito et al. (1973) treated male dd mice (12/group) with Kanechlors 500, 400 and 300 each at dietary levels of 100, 250 or 500 ppm for 32 weeks. The group fed 500 ppm of Kanechlor 500 had a 41.7% incidence of hepatocellular carcinomas and a 58.3% incidence of nodular hyperplasia. Hepatocellular carcinomas and nodular hyperplasia were not observed in mice fed 100 or 250 ppm of Kanechlor 500, nor among those fed Kanechlors 400 or 300 at any concentrations.

Schaeffer et al. (1984) fed male Wistar rats diets containing 100 ppm of the PCB mixtures Clophen A 30 (30% chlorine by weight) or Clophen A 60 (60% chlorine by weight) for 800 days. The PCB mixtures were reported to be free of furans. Clophen A 30 was administered to 152 rats, Clophen A 60 to 141 rats, and 139 rats received a standard diet. Mortality and histologic lesions were reported for animals necropsied during each 100-day interval for all

three groups. Of the animals that survived the 800-day treatment period, 1/53 rats (2%) in the control group, 3/87 (3%) in the Clophen A 30 group and 52/85 (61%) in the Clophen A 60 group had developed hepatocellular carcinoma. The incidence in the Clophen A 60 group was significantly elevated in comparison to the control group. Neoplastic nodules were reported in 2/53 control, 35/87 Clophen A 30, and 34/85 Clophen A 60-treated animals. The incidence of nodules was significantly increased in both treatment groups in comparison to the control group. Neoplastic liver nodules and hepatocellular carcinomas appeared earlier and at higher incidence in the Clophen A 60 group relative to the Clophen A 30 group. The authors interpreted the results as indicative of a carcinogenic effect related to the degree of chlorination of the PCB mixture. The authors also suggested that these findings support those of others, including Ito et al. (1973) and Kimbrough et al. (1975), in which hepatocellular carcinomas were produced by more highly chlorinated mixtures.

Kimbrough and Linder (1974) dosed groups of 50 male BALB/cJ mice (a strain with a low spontaneous incidence of hepatoma) with Aroclor 1254 at 300 ppm in the diet for 11 months or 6 months, followed by a 5-month recovery period. Two groups of 50 mice were fed a control diet for 11 months. The incidence of hepatomas in survivors fed Aroclor 1254 for 11 months was 10/22. One hepatoma was observed in the 24 survivors fed Aroclor 1254 for 6 months.

o SUPPORTING DATA :

Most genotoxicity assays of PCBs have been negative. The majority of microbial assays of PCB mixtures and various congeners showed no evidence of mutagenic effects (Schoeny et al., 1979; Schoeny, 1982; Wyndham et al., 1976).

Of various tests on the clastogenic effect of PCBs (Heddle and Bruce, 1977; Green et al., 1975), only Peakall et al. (1972) reported results indicative of a possible clastogenic action by PCBs in dove embryos.

Chlorinated dibenzofurans (CDFs), known contaminants of PCBs, and chlorinated dibenzodioxins (CDDs) are structurally related to and produce certain biologic effects similar to those of PCB congeners. While the CDDs are known to be carcinogenic, the carcinogenicity of CDFs is still under evaluation.

CARO -

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : hepatocellular carcinomas in three strains of rats and two strains of mice and inadequate yet suggestive evidence of excess risk of liver cancer in humans by ingestion and inhalation or dermal contact.
- o ORAL SLOPE FACTOR : 7.7/mg/kg/day
- o DRINKING WATER UNIT RISK : 2.2E-4/ug/L
- o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
- o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	5E-1 ug/L
E-5 (1 in 100,000)	5E-2 ug/L
E-6 (1 in 1,000,000)	5E-3 ug/L

o ORAL DOSE-RESPONSE DATA :

Tumor Type -- trabecular carcinoma/adenocarcinoma, neoplastic nodule
 Test Animals -- rat/Sprague-Dawley, female
 Route -- diet
 Reference -- Norback and Weltman, 1985

Administered Dose (mg/kg)/day (TWA)	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
0	0	1/49
3.45	0.59	45/47

o ADDITIONAL COMMENTS :

Human equivalent dosage assumes a TWA daily dose of 3.45 mg/kg/day. This reflects the dosing schedule of 5 mg/kg/day (assuming the rat consumes an amount equal to 5% of its bw/day) for the first 16 months, 2.5 mg/kg/day for the next 8 months, and no dose for the last 5 months.

A slope factor of 3.9/mg/kg/day was based on data from the Kimbrough et al. (1975) study of female Sherman rats fed Aroclor 1260. The estimate based on the data of Norback and Weltman (1985) is preferred because Sprague-Dawley rats are known to have low incidence of spontaneous hepatocellular neoplasms. Moreover, the latter study spanned the natural life of the animal, and concurrent morphologic liver studies showed the sequential progression of liver lesions to hepatocellular carcinomas.

Although it is known that PCB congeners vary greatly as to their potency in producing biological effects, for purposes of this carcinogenicity assessment Aroclor 1260 is intended to be representative of all PCB mixtures. There is some evidence that mixtures containing more highly chlorinated biphenyls are more potent inducers of hepatocellular carcinoma in rats than mixtures containing less chlorine by weight (reviewed in Kimbrough, 1987 and Schaeffer et al., 1984).

The unit risk should not be used if the water concentration exceeds 50 ug/L, since above this concentration the slope factor may differ from that stated.

o DISCUSSION OF CONFIDENCE :

The Norback and Weltman study used an adequate number of animals, observed for their normal lifespan. Only one non-zero test dose was used. A second risk estimate was also calculated based on the numbers of malignant tumors alone, as called for in the EPA's guidelines for carcinogen risk assessment. The slope factor thus derived is 5.7/mg/kg/day, which is 26% less than that derived using combined malignant tumors and neoplastic nodules. This risk

estimate is supported by one based on data of Kimbrough et al. (1975).

PCB mixtures in drinking water may not be the same as the mixtures introduced or used for testing carcinogenicity in animals.

CARI - NO DATA

CARDR-

o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1988

The 1988 Drinking Water Criteria Document for PCBs has received OHEA review.
DOCUMENT

o REVIEW DATES : 04/22/87

o VERIFICATION DATE : 04/22/87

o EPA CONTACTS :

Charli Hiremath / OHEA -- (202)260-5725

Debdas Mukerjee / OHEA -- (513)569-7572

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 7.9E-5 ug/L

Fish Consumption Only: 7.9E-6 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. Since zero, however, may not be attainable at this time, the recommended criteria represents an E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 2.0E+0 ug/L
Chronic -- 1.4E-2 ug/L

Marine:

Acute -- 1.0E+1 ug/L
Chronic -- 3.0E-2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0.0 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- The MCLG for polychlorinated biphenyls is zero based on the evidence of carcinogenic potential (classification B2).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.0005 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- The MCL is based on a PQL of 0.0005 mg/L and is associated with a maximum lifetime individual risk of E-4.

Monitoring requirements -- All systems monitored initially for four consecutive quarters every three years; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Microextraction/gas chromatography (EPA 505); electron capture detector (EPA 508); perchlorination/gas chromatography (EPA 508A). PQL= 0.0005 mg/L.

Best available technology -- Granular activated carbon

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ for polychlorinated biphenyls is based on aquatic toxicity. The available data indicate that the 96-Hour Median Threshold Limit is less than 0.1 ppm, which corresponds to an RQ of 1 pound.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

___IV.E.1. TSCA, SECTION 6

Status -- Final Rule (1988)

Discussion -- Prohibits the manufacture, processing, distribution in commerce, or the use of PCBs other than in a "totally enclosed manner" unless specifically exempted by the EPA. Reporting, disposal and record-keeping requirements. Advance notice of proposed rulemaking [56 FR 26738, (06/10/91)] to amend TSCA PCB disposal regulations.

Reference -- 52 FR 27322 (07/19/88); 55 FR 21033 (05/22/90)

EPA Contact -- Chemical Control Division / OTS
(202) 260-3749 / FTS 260-3749

OREF - None

IREF - None

CREF - Amano, M., K. Yagi, H. Nakajima, R. Takehara, H. Sakai and G. Umeda. 1984. Statistical observations about the causes of the death of patients with oil poisoning. Japan Hygiene. 39(1): 1-5.

CREF - Bahn, A.K., I. Rosenwaike, N. Herrmann, P. Grover, J. Stellman and K. O'Leary. 1976. Melanoma after exposure to PCB's. New Engl. J. Med. 295: 450.

CREF - Bahn, A.K., P. Grover, I. Rosenwaike, K. O'Leary and J. Stellman. 1977. Reply to letter from C. Lawrence entitled, "PCB? and melanoma". New Engl. J. Med. 296: 108.

CREF - Bertazzi, P.A., L. Riboldi, A. Pesatori, L. Radice and C. Zacchetti.

1987. Cancer mortality of capacitor manufacturing workers. *Am. J. Ind. Med.* 11(2): 165-176.
- CREF - Brown, D.P. 1987. Mortality of workers exposed to polychlorinated biphenyls - An update. *Arch. Environ. Health.* 42(6): 333-339.
- CREF - Brown, D.P. and M. Jones. 1981. Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. *Arch. Environ. Health.* 36(3): 120-129.
- CREF - Green, S., J.V. Carr, K.A. Palmer and E.J. Oswald. 1975. Lack of cytogenetic effects in bone marrow and spermatogonial[sic] cells in rats treated with polychlorinated biphenyls (Aroclors 1242 and 1254). *Bull. Environ. Contam. Toxicol.* 13(1): 14-22.
- CREF - Heddle, J.A. and W.R. Bruce. 1977. Comparison of tests for mutagenicity or carcinogenicity using assays for sperm abnormalities, formation of micronuclei and mutations in *Salmonella*. In: *Origins of Human Cancer*, H.H. Hiatt et al., Ed. Cold Spring Harbor Conf. Cell Prolif., Cold Spring Harbor Lab., Cold Spring Harbor, NY. 4: 1549-1557.
- CREF - Ito, N., H. Nagasaki, M. Arai, S. Makiura, S. Sugihara and K. Hirao. 1973. Histopathologic studies on liver tumorigenesis induced in mice by technical polychlorinated biphenyls and its promoting effect on liver tumors induced by benzene hexachloride. *J. Natl. Cancer Inst.* 51(5): 1637-1646.
- CREF - Ito, N., H. Nagasaki, S. Makiura and M. Arai. 1974. Histopathological studies on liver tumorigenesis in rats treated with polychlorinated biphenyls. *Gann.* 65: 545-549.
- CREF - Kimbrough, R.D. 1987. Human health effects of polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs). *Ann. Rev. Pharmacol. Toxicol.* 27: 87-111.
- CREF - Kimbrough, R.D. and R.E. Linder. 1974. Induction of adenofibrosis and hepatomas in the liver of BALB/cJ mice by polychlorinated biphenyls (Aroclor 1254). *J. Natl. Cancer Inst.* 53(2): 547-552.
- CREF - Kimbrough, R.D., R.A. Squire, R.E. Linder, J.D. Strandberg, R.J. Montali and V.W. Burse. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. *J. Natl. Cancer Inst.* 55(6): 1453- 1459.
- CREF - Kimura, N.T. and T. Baba. 1973. Neoplastic changes in the rat liver induced by polychlorinated biphenyl. *Gann.* 64: 105-108.
- CREF - NCI (National Cancer Institute). 1978. Bioassay of Aroclor (trademark) 1254 for possible carcinogenicity. CAS No. 27323-18-8. NCI Carcinogenesis Tech. Rep. Ser. No. 38.
- CREF - NIOSH (National Institute for Occupational Safety and Health). 1977. Criteria for a Recommended Standard . . . Occupational Exposure to Polychlorinated Biphenyls (PCBs). U.S. DHEW, PHS, CDC, Rockville, Md. Publ. No. 77-225.
- CREF - Norback, D.H. and R.H. Weltman. 1985. Polychlorinated biphenyl induction of hepatocellular carcinoma in the Sprague-Dawley rat. *Environ. Health Perspect.* 60: 97-105.
- CREF - Peakall, D.B., J.L. Lincer and S.E. Bloom. 1972. Embryonic mortality and chromosomal alterations caused by Aroclor 1254 in ring doves. *Environ. Health Perspect.* 1: 103-104.
- CREF - Schaeffer, E., H. Greim and W. Goessner. 1984. Pathology of chronic polychlorinated biphenyl (PCB) feeding in rats. *Toxicol. Appl. Pharmacol.* 75: 278-288.
- CREF - Schoeny, R. 1982. Mutagenicity testing of chlorinated biphenyls and chlorinated dibenzofurans. *Mutat. Res.* 101: 45-56.

CREF - Schoeny, R.S., C.C. Smith and J.C. Loper. 1979. Non-mutagenicity for Salmonella of the chlorinated hydrocarbons Aroclor 1254, 1,2,4-trichlorobenzene, mirex and kepone. Mutat. Res. 68: 125-132.

CREF - U.S. EPA. 1988. Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

CREF - Wyndham, C., J. Devenish and S. Safe. 1976. The in vitro metabolism, macromolecular binding and bacterial mutagenicity of 4-chlorobiphenyl, a model PCB substrate. Res. Commun. Chem. Pathol. Pharmacol. 15: 563-570.

HAREF- None

gamma-Chlordane

1 - IRIS
 NAME - Chlordane
 RN - 57-74-9
 IRSN - 139
 DATE - 930701
 UPDT - 07/01/93, 6 fields
 STAT - Oral RfD Assessment (RDO) on-line 07/01/89
 STAT - Inhalation RfC Assessment (RDI) pending
 STAT - Carcinogenicity Assessment (CAR) on-line 07/01/93
 STAT - Drinking Water Health Advisories (DWHA) on-line 08/01/90
 STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
 IRH - 09/30/87 CAR Carcinogenicity section added
 IRH - 03/01/88 RDO Dose conversion clarified
 IRH - 03/01/88 RDO Text clarified in paragraph 3
 IRH - 03/01/88 CAREV Basis for classification clarified
 IRH - 03/01/88 HADV Health Advisory added
 IRH - 04/01/89 RDO Withdrawn; new RfD verified (in preparation)
 IRH - 06/01/89 RDO Revised oral RfD summary added
 IRH - 06/01/89 REFS Bibliography on-line
 IRH - 07/01/89 RDO Reference clarified in paragraph 2
 IRH - 07/01/89 CAR Velsicol (1983) references clarified
 IRH - 07/01/89 CREF Carcinogen references added
 IRH - 03/01/90 RDI Inhalation RfD now under review
 IRH - 08/01/90 HALIF DWEL changed reflecting change in RfD
 IRH - 08/01/90 HADR Primary contact changed
 IRH - 08/01/90 RCRA EPA contact changed
 IRH - 01/01/91 CAR Text edited
 IRH - 01/01/91 CARL Inhalation slope factor removed (global change)
 IRH - 01/01/92 EXSR Regulatory actions updated
 IRH - 07/01/93 CARDR Secondary contact's phone number changed
 RLEN - 28857
 SY - Belt
 SY - CD 68
 SY - Chlordane
 SY - Chlorindan
 SY - Chlor Kil
 SY - Corodan
 SY - Dowchlor
 SY - ENT 9,932
 SY - HCS 3260
 SY - Kypchlor
 SY - M 140
 SY - M 410
 SY - 4,7-Methanoindan, 1,2,4,5,6,7,8,8-Octachloro-3a,4,7,7a-Tetrahydro-
 SY - 4,7-Methano-1H-Indene,
 1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-Hexahydro-
 SY - NCI-C00099
 SY - Niran
 SY - Octachlorodihydrodicyclopentadiene
 SY - 1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-Hexahydro-4,7-Methano-indene
 SY - 1,2,4,5,6,7,8,8-Octachloro-3a,4,7,7a-Hexahydro-4,7-Methylene Indane
 SY - Octachloro-4,7-Methanohydroindane
 SY - Octachloro-4,7-Methanotetrahydroindane
 SY - Octa-Klor
 SY - Oktaterr

SY - Ortho-Klor
SY - Synklor
SY - TAT Chlor 4
SY - Topiclor
SY - Toxichlor
SY - Velsicol 1068

RDO -

o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Regional liver hypertrophy in females	NOEL: 1 ppm (0.055 mg/kg/day)	1000	1	6E-5 mg/kg/day

30-Month Rat Feeding Study LEL: 5 ppm
(0.273 mg/kg/day)

Velsicol Chemical Co.,
1983a

*Conversion Factors: Actual dose tested

o ORAL RFD STUDIES :

Velsicol Chemical Company. 1983a. MRID No. 00138591, 00144313. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Charles River Fischer 344 rats (80/sex/dose) were fed technical chlordane at dietary levels of 0, 1, 5, and 25 ppm for 130 weeks. Body weight, food consumption, and water uptake were monitored at regular intervals. Clinical laboratory studies were performed and organ weights measured on eight animals/sex/group at weeks 26 and 52, and on all survivors at week 130. Gross and microscopic pathology were performed on all tissues. Daily dose level of 0.045, 0.229, and 1.175 mg/kg/day for males and 0.055, 0.273, and 1.409 mg/kg/day for females for the 1, 5, and 25 ppm treatment groups, respectively, were calculated from food consumption and body weight data.

Following the submission of a 30-month chronic feeding/oncogenicity study in Fischer 344 rats, the Agency reviews by the Office of Pesticides Programs and the Cancer Assessment Group of these data indicated that male rats at the highest dosage exhibited an increase in liver tumors (ICF Clement, 1987). The registrant, Velsicol Chemical Company, subsequently convened the Pathology Working Group to reevaluate the slides of livers of the chlordane-treated rats reported in MRID No. 00138591. It was concluded that liver lesions had not occurred in male rats and that 25 ppm (0.1175 mg/kg/day) was the NOEL for males. Liver lesions (hypertrophy), however, had occurred in female rats at 5 ppm (0.273 mg/kg/day), which was considered an LEL. Therefore an NOEL of 1 ppm (0.055 mg/kg/day) (LDT) was established for female rats.

o ORAL RFD UNCERTAINTY :

UF - An uncertainty factor of 100 was used to account for the inter- and intraspecies differences. An additional UF of 10 was used to account for the lack of an adequate reproduction study and adequate chronic study in a second

mammalian species, and the generally inadequate sensitive endpoints studied in existing studies, particularly since chlordane is known to bioaccumulate over a chronic duration.

o ORAL RFD MODIFYING FACTOR :

MF – None

o ORAL RFD COMMENTS :

Data Considered for Establishing the RfD

1) 30-Month Feeding (oncogenic) - rat: Principal study - see previous description; core grade minimum

2) 24-Month Chronic Toxicity - mouse: NOEL=1 ppm (0.15 mg/kg/day); LEL=5 ppm (0.75 mg/kg/day) (hepatocellular swelling and necrosis in males; hepatocyte swelling in males, and increased live weight in males and females); At 12.5 ppm (1.875 mg/kg/day) (HDT); core grade minimum (Velsicol Chemical Co., 1983b)

Data Gap(s): Chronic Dog Feeding Study, Rat Reproduction Study, Rat Teratology Study, Rabbit Teratology Study

o ORAL RFD CONFIDENCE :

Study – Medium
Data Base – Low
RfD – Low

The critical study is of adequate quality and is given a medium rating. The data base is given a low confidence rating because of 1) the lack of an adequate reproduction study and adequate chronic study in a second mammalian species and 2) inadequate sensitive endpoints studied in existing studies, particularly since chlordane is known to bioaccumulate over a chronic duration. Low confidence in the RfD follows.

o ORAL RFD SOURCE DOCUMENT :

Source Document – This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation – Pesticide Registration Standard, November 1986; Pesticide Registration Files

o REVIEW DATES : 12/18/85, 03/22/89
o VERIFICATION DATE : 03/22/89
o EPA CONTACTS :

George Ghali / OPP – (703)557-7490

William Burnam / OPP – (703)557-7491

RDI -

o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

o REVIEW DATES : 02/22/90

CAREV-

o CLASSIFICATION : B2; probable human carcinogen

o BASIS FOR CLASSIFICATION : Sufficient evidence in studies in which benign and malignant liver tumors were induced in four strains of mice of both sexes and in F344 male rats; structurally related to other liver carcinogens

o HUMAN CARCINOGENICITY DATA :

Inadequate. There were 11 case reports involving central nervous system effects, blood dyscrasias and neuroblastomas in children with pre-/postnatal exposure to chlordane and heptachlor (Infante et al., 1978). As no other information was available, no conclusions can be drawn.

There were three epidemiologic studies of workers exposed to chlordane and/or heptachlor. One study of pesticide applicators was considered inadequate in sample size and duration of follow-up. This study showed marginal statistically significant increased mortality from bladder cancer (3 observed) (Wang and McMahon, 1979a). The other two studies were of pesticide manufacturing workers. Neither of them showed any statistically significantly increased cancer mortality (Wang and McMahon, 1979b; Ditraglia et al., 1981). Both these populations also had confounding exposures from other chemicals.

o ANIMAL CARCINOGENICITY DATA :

Sufficient. Chlordane has been studied in four mouse and four rat long-term carcinogenesis bioassays. Dose-related incidences of liver carcinoma constitute the major finding in mice. Becker and Sell (1979) tested chlordane (90:10 mixture of chlordane to heptachlor) in C57B1/6N mice, a strain historically known not to develop spontaneous liver tumors. An unspecified number of mice were fed chlordane at 0, 25 and 50 ppm (0, 3.57, 7.14 mg/kg bw) for 18 months. None of the controls developed tumors or nodular lesions of the liver. Twenty-seven percent (16 mice) of the surviving treated mice developed primary hepatocellular carcinomas. Velsicol (1973) fed groups of 100 male and 100 female CD-1 mice diets with 0, 5, 25 or 50 ppm analytical grade chlordane for 18 months. A significant ($p < 0.01$) dose-related increase in nodular hyperplasias in the liver of male and female mice was reported at the two highest dose levels. A histological review by Reuber (U.S. EPA, 1985) reported a high incidence ($p < 0.01$) of hepatic carcinomas instead of hyperplastic nodules at 25 and 50 ppm.

A dose-related increase ($p < 0.001$ after lifetable adjustment) of hepatocellular carcinomas was also observed in both sexes of B6C3F1 mice (NCI, 1977). Male and female mice were fed technical-grade chlordane (purity = 94.8%) at TWA concentrations (TWAC) of 29.9 and 56.2 ppm and 30.1 and 63.8 ppm, respectively, for 80 weeks. In this study there were individual matched controls for the low and high dose groups. ICR male mice developed

hepatocellular adenomas and hemangiomas when fed 12.5 ppm chlordane for 24 months. No tumors were observed in the female mice when tested at the same concentrations: 0, 1, 5, and 12.5 ppm (Velsicol, 1983a).

Velsicol (1983b) reported a long-term (130 weeks) carcinogenesis bioassay on 80 male and 80 female F344 rats fed concentrations of 0, 1, 5, and 25 ppm chlordane. A significant increase in adenomas of the liver was observed in male rats receiving 25 ppm. Although no tumors were observed in female rats, hepatocellular swelling was significantly increased at 25 ppm. The NCI (1977) reported a significant increase ($p < 0.05$) of neoplastic nodules of the liver in low-dose Osborne-Mendel female rats (TWAC of 120.8 ppm) but not in the high-dose group (TWAC of 241.5 ppm). No tumor incidence was reported for the males fed TWAC of 203.5 and 407 ppm. Loss of body weight and a dose-related increase in mortality was observed in all treated groups. High mortality and reduced growth rates in Osborne-Mendel rats was also observed by Ingle (1952) when the rats were exposed to 150 and 300 ppm chlordane but not at 5, 10, and 30 ppm. No treatment-related incidence of tumors was reported. Significantly enlarged livers and liver lesions were found in male and female albino rats fed chlordane at greater than or equal to 80 ppm (Ambrose et al., 1953a,b). No treatment-related increase in tumors was found, but the study duration (400 days) was short.

o SUPPORTING DATA :

Gene mutation assays indicate that chlordane is not mutagenic in bacteria (Wildeman and Nazar, 1982; Probst et al., 1981; Gentile et al., 1982). Positive results have been reported in Chinese hamster lung V79 cells and mouse lymphoma L5178Y cells with and without exogenous metabolism, as well as in plant assays. Chlordane did not induce DNA repair in bacteria, rodent hepatocytes (Maslansky and Williams, 1981), or human lymphoid cells (Sobti et al., 1983). It is a genotoxin in yeast (Gentile et al., 1982; Chambers and Dutta, 1976), human fibroblasts (Ahmed et al., 1977), and fish (Vigfusson et al., 1983).

Five compounds structurally related to chlordane (aldrin, dieldrin, heptachlor, heptachlor epoxide, and chlorendic acid) have produced liver tumors in mice. Chlorendic acid has also produced liver tumors in rats.

CARO -

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : Sufficient evidence in studies in which benign and malignant liver tumors were induced in four strains of mice of both sexes and in F344 male rats; structurally related to other liver carcinogens
- o ORAL SLOPE FACTOR : $1.3E+0$ per (mg/kg)/day
- o DRINKING WATER UNIT RISK : $3.7E-5$ per (ug/L)
- o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
- o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	3E+0 ug/L
E-5 (1 in 100,000)	3E-1 ug/L
E-6 (1 in 1,000,000)	3E-2 ug/L

o ORAL DOSE-RESPONSE DATA :

Tumor Type – hepatocellular carcinoma
 Test Animals – mouse/CD-1 (Velsicol); mouse/B6C3F1 (NCI)
 Route – diet
 Reference – Velsicol, 1973; NCI, 1977

Administered Dose (ppm)	Human Equivalent Dose (mg/kg-day)	Tumor Incidence	Reference
female			
0	0.000	0/45	Velsicol, 1973
5	0.052	0/61	
25	0.260	32/50	
50	0.520	26/37	
male			
0	0.000	3/33	Velsicol, 1973
5	0.052	5/55	
25	0.260	41/52	
50	0.520	32/39	
male			
0	0.00	2/18	NCI, 1977
29.9	0.31	16/48	
56.2	0.58	43/49	
female			
0	0.00	0/19	NCI, 1977
30.1	0.31	3/47	
63.8	0.66	34/49	

o ADDITIONAL COMMENTS :

Four data sets for mice and one data set for rats showed a significant increase in liver tumors; namely hepatocellular carcinomas in mice (NCI, 1977; Velsicol, 1973) and hepatocellular adenomas in rats (Velsicol, 1983a). The quantitative estimate is based on the geometric mean from the four mouse data sets as mice were the more sensitive species tested and as risk estimates for a similar compound (heptachlor) were similarly derived from mouse tumor data. The slope factors for the data sets are these: 2.98 per (mg/kg)/day for CD-1 female mice, 4.74 per (mg/kg)/day for CD-1 male mice, 0.76 per (mg/kg)/day for B6C3F1 male mice, and 0.25 per (mg/kg)/day for B6C3F1 female mice. Low and high dose groups in the NCI (1977) study had individual matched controls.

The unit risk should not be used if the water concentration exceeds 300 ug/L, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

Liver carcinomas were induced in mice of both sexes in two studies. An

adequate number of animals was observed, and dose-response effects were reported in all studies. The geometric mean of slope factors (0.25 to 4.74 per (mg/kg)/day for the most sensitive species is consistent with that derived from rat data (1.11/mg/kg/day).

CARI -

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : Sufficient evidence in studies in which benign and malignant liver tumors were induced in four strains of mice of both sexes and in F344 male rats; structurally related to other liver carcinogens
- o INHALATION UNIT RISK : $3.7E-4$ per (ug/cu.m)
- o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
- o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	$3E-1$ ug/cu.m
E-5 (1 in 100,000)	$3E-2$ ug/cu.m
E-6 (1 in 1,000,000)	$3E-3$ ug/cu.m

o INHALATION DOSE-RESPONSE DATA :

The inhalation risk estimates were calculated from the oral data presented in CARO.

o ADDITIONAL COMMENTS :

The unit risk should not be used if the air concentration exceeds 30 ug/cu.m, above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

See CARO.

CARDR-

o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1986, 1985

The values in the 1986 Carcinogenicity Assessment for Chlordane and Heptachlor/Heptachlor Epoxide have been reviewed by the Carcinogen Assessment Group.

DOCUMENT

o REVIEW DATES : 04/01/87
o VERIFICATION DATE : 04/01/87
o EPA CONTACTS :

Dharm V. Singh / OHEA – (202)260-5958

Jim Cogliano / OHEA – (202)260-3814

HAONE-

Appropriate data for calculating a One-day HA are not available. It is recommended that the Ten-day HA of 0.06 mg/L be used as the One-day HA.

HATEN-

Ten-day HA – 6E-2 mg/L

LOAEL – 6.25 mg/kg/day

UF – 1000 (allows for interspecies and intrahuman variability with the use of a LOAEL from an animal study)

Assumptions – 1 L/day water consumption for a 10-kg child

Principal Study – Ambrose et al., 1953

The toxic effects in rats resulting from daily gastric intubation of chlordane at doses of 6.25, 12.5, 25.0, 50.0, 100.0, or 200 mg/kg for 15 days were histologic changes in the liver of the treated animals at all dose levels and central nervous system effects at higher dose levels. Only minimal histopathologic changes characterized by the presence of abnormal intracytoplasmic bodies of various diameters were evident at the lowest dose level (6.25 mg/kg). That dose level was identified as the LOAEL in this study.

HALTC-

Appropriate data for calculating a Longer-term HA are not available. It is recommended that the modified DWEL (adjusted for a 10-kg child) of 0.5 ug/L be used as the Longer-term HA.

HALTA-

Appropriate data for calculating a Longer-term HA are not available. It is recommended that the DWEL of 2 ug/L be used as the Longer-term HA for the 70-kg adult.

HALIF-

Drinking Water Equivalent Level (DWEL) – 2E-3 mg/L

Assumptions – 2 L/day water consumption for a 70-kg adult

RfD Verification Date = 03/22/89 (see RDO)

Lifetime HA -- None

Chlordane is considered to be a probable human carcinogen. Refer to CAR for information on the carcinogenicity of this substance.

Principal Study (DWEL) -- Velsicol Chemical Corporation, 1983 (This study was used in the derivation of the chronic oral RfD; see RDO)

OLEP -

No data available

ALAB -

Determination of chlordane is by a liquid-liquid extraction gas chromatographic procedure.

TREAT-

Treatment technologies which are capable of removing chlordane from drinking water include adsorption by granular or powdered activated carbon and air stripping.

HADR -

o HEALTH ADVISORY SOURCE :

U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Chlordane. Office of Drinking Water, Washington, DC.
DOCUMENT

o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1985.

Public review of HAs following notification of availability in October, 1985.

Scientific Advisory Panel review of HAs in January, 1986.

o EPA DRINKING WATER CONTACT :

Jennifer Orme Zavaleta / OST -- (202)260-7586

Edward V. Ohanian / OST -- (202)260-7571

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 4.6E-4 ug/L

Fish Consumption Only: 4.8E-4 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be obtainable at this time, so the recommended criteria represents an E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 2.4 E+ 0 ug/L (at any time)
Chronic -- 4.3 E- 3 ug/L (24-hour average)

Marine:

Acute -- 9.0 E-2 ug/L (at any time)
Chronic -- 4.0 E-3 ug/L (24-hour average)

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion — An MCLG of 0 mg/L for chlordane is promulgated based upon carcinogenic effects (B2).

Reference — 56 FR 3526 (01/30/91)

EPA Contact — Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value — 0.002 mg/L (Final, 1991)

Considers technological or economic feasibility? — YES

Discussion — EPA has set a MCL equal to the PQL of 0.002, which is associated with a lifetime individual risk of 1.5×10^{-4} .

Monitoring requirements — All systems monitored for four consecutive quarters every three years; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology — Microextraction/gas chromatography (EPA 505); electron-capture/gas chromatography (EPA 508); gas chromatography/mass spectrometry (EPA 525): PQL= 0.002 mg/L.

Best available technology — Granular activated carbon

Reference — 56 FR 3526 (01/30/91)

EPA Contact — Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

SMCL - NO DATA

FISTD-

Status — Issued (1986)

Reference -- Chlordane Pesticide Registration Standard. December, 1986 (NTIS No. PB87-175816).

EPA Contact -- Registration Branch, OPP / (703)557-7760 / FTS 557-7760

FIREV-

Action -- Cancellation of many termiticide products (1988)

Considers technological or economic feasibility? -- YES

Summary of regulatory action -- 43 FR 12372 (03/24/78) - Cancellation of all but termiticide use; under the provisions of the Administrator's acceptance of the settlement plan to phase out certain uses of chlordane, most registered products containing chlordane were effectively canceled or the applications for registration were denied by 12/31/80. A summary of those uses not affected by this settlement, or a previous suspension, follows: 1) subsurface ground insertion for termite control (clarified by 40 FR 30522, July 21, 1975, to apply to the use of emulsifiable or oil concentrate formulations for controlling subterranean termites on structural sites such as buildings, houses, barns, and sheds, using current control practices), 2) dipping of nonfood roots and tops. 52 FR 42145 (11/03/87) - Negotiated agreement on termiticide use. The agreement (order) accepted voluntary cancellations of the registration of certain pesticide products and imposed limitations on the continued sale, distribution, and use of existing stocks of such products/ criterion of concern: oncogenicity,

Reference -- 43 FR 12372 (03/24/78); 52 FR 42145 (11/03/87); 53 FR 11798 (04/08/88)

EPA Contact -- Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for chlordane is 1 pound, based on aquatic toxicity, as established under CWA Section 311 (40 CFR 117.3). Available data indicate the aquatic 96-hour Median Threshold Limit for chlordane is less than 0.1 ppm. This corresponds to an RQ of 1 pound. Chlordane has also been found to bioaccumulate in the tissues of aquatic and marine organisms, and has the potential to concentrate in the food chain.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline

(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - ICF-Clement. 1987. MRID No. 40433701. Available from EPA. Write to FOI, EPA, Washington DC 20460.

OREF - Velsicol Chemical Co. 1983a. MRID No. 00138591, 00144313. Available from EPA. Write to FOI, EPA, Washington DC 20460.

OREF - Velsicol Chemical Co. 1983b. MRID No. 00144312. Available from EPA. Write to FOI, EPA, Washington DC 20460.

IREF - None

CREF - Ahmed, F.E., R.W. Hart and N.J. Lewis. 1977. Pesticide induced DNA damage and its repair in cultured human cells. *Mutat. Res.* 42: 161-174.

CREF - Ambrose, A.M., H.E. Christensen, D.J. Robbins and L.J. Rather. 1953a. Toxi- cological and pharmacological studies on chlordane. *Arch. Ind. Hyg. Occup. Med.* 7: 197-210.

CREF - Ambrose, A.M., H.E. Christensen and D.J. Robbins. 1953b. Pharmacological observations on chlordane. *Fed. Proceed.* 12: 298. (Abstract #982)

CREF - Becker, F.F. and S. Sell. 1979. Fetoprotein levels and hepatic alterations during chemical carcinogenesis in C57BL/6N mice. *Cancer Res.* 39: 3491-3494.

CREF - Chambers, D. and S.K. Dutta. 1976. Mutagenic tests of chlordane on different microbial tester strains. *Genetics.* 83: s13. (Abstract)

CREF - Ditraglia, D., D.P. Brown, T. Namekata and N. Iverson. 1981. Mortality study of workers employed at organochlorine pesticide manufacturing plants. *Scand. J. Work Environ. Health.* 7(4): 140-146.

CREF - Gentile, J.M., G.J. Gentile, J. Bultman, R. Sechriest, E.D. Wagner and M.J. Plewa. 1982. An evaluation of the genotoxic properties of insecticides following plant and animal activation. *Mutat. Res.* 101: 19-29.

CREF - Infante, P.F., S.S. Epstein and W.A. Newton. 1978. Blooddyscrasis and childhood tumors and exposure to chlordane and heptachlor. *Scand. J. Work Environ. Health.* 4: 137-150.

- CREF - Ingle, L. 1952. Chronic oral toxicity of chlordane to rats. Arch. Ind. Hyg. Occup. Med. 6: 357-367.
- CREF - Maslansky, C.J. and G.M. Williams. 1981. Evidence for an epigenetic mode of action in organochlorine pesticide hepatocarcinogenicity: A lack of genotoxicity in rat, mouse, and hamster hepatocytes. J. Toxicol. Environ. Health. 8: 121-130.
- CREF - NCI (National Cancer Institute). 1977. Bioassay of Chlordane for possible Carcinogenicity. NCI Carcinogenesis Tech. Rep. Ser. No. 8. U.S. DHEW Publ. No. (NIH) 77-808. Bethesda, MD.
- CREF - Probst, G.S., R.E. McMahon, L.E. Hill, C.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 compounds. Environ. Mutagen. 3: 11-31.
- CREF - Sobti, R.C., A. Krishan and J. Davies. 1983. Cytokinetic and cytogenetic effect of agricultural chemicals on human lymphoid cells in vitro. Arch. Toxicol. 52: 221-231.
- CREF - U.S. EPA. 1985. Hearing Files on Chlordane, Heptachlor Suspension (unpublished draft). Available for inspection at U.S. EPA, Washington, DC.
- CREF - U.S. EPA. 1986. Carcinogenicity Assessment of Chlordane and Heptachlor/Heptachlor Epoxide. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC.
- CREF - Velsicol Chemical Corporation. 1973. MRID No. 00067568. Available from EPA. Write to FOI, EPA, Washington, DC. 20460.
- CREF - Velsicol Chemical Corporation. 1983a. MRID No. 00144312, 00132566. Available from EPA. Write to FOI, EPA, Washington, DC. 20460.
- CREF - Velsicol Chemical Corporation. 1983b. MRID No. 00138591. Available from EPA. Write to FOI, EPA, Washington, DC. 20460.
- CREF - Vigfusson, N.V., E.R. Vyse, C.A. Pernsteiner and R.J. Dawson. 1983. In vivo induction of sister-chromatid exchange in Umbra limi by the insecticides endrin, chlordane, diazinon and guthion. Mutat. Res. 118: 61-68.
- CREF - Wang, H.H. and B. MacMahon. 1979a. Mortality of workers employed in the manufacture of chlordane and heptachlor. J. Occup. Med. 21(11): 745-748.
- CREF - Wang, H.H. and B. MacMahon. 1979b. Mortality of pesticide applicators. J. Occup. Med. 21(11): 741-744.
- CREF - Wildeman, A.G. and R.N. Nazar. 1982. Significance of plant metabolism in the mutagenicity and toxicity of pesticides. Can. J. Genet. Cytol. 24: 437-449.
- HAREF - Ambrose, A.M., H.E. Christensen, D.J. Robbins and L.J. Rather. 1953. Toxicological and pharmacological studies on chlordane. Arch. Ind. Hyg. Occup. Med. 7: 197-210.
- HAREF - U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Chlordane. Office of Drinking Water, Washington, DC.
- HAREF - Velsicol Chemical Corp. 1983. MRID No. 00138591. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

2-Nitroaniline

1 - IRIS
NAME - 2-Nitroaniline
RN - 88-74-4
IRSN - 627
DATE - 920807
UPDT - 08/07/92, 52 fields
STAT - Oral RfD Assessment (RDO) pending 08/01/92
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
IRH - 08/01/92 RDO Oral RfD now under review
RLEN - 1240
SY - Benzenamine, 2-nitro-
SY - O-NITROANILINE
SY - 2-NITROANILINE
SY - AI3-02916
SY - Aniline, o-nitro-
SY - AZOENE FAST ORANGE GR BASE
SY - AZOENE FAST ORANGE GR SALT
SY - AZOFIX ORANGE GR SALT
SY - AZOGENE FAST ORANGE GR
SY - AZOIC DIAZO COMPONENT 6
SY - BRENTAMINE FAST ORANGE GR BASE
SY - BRENTAMINE FAST ORANGE GR SALT
SY - C.I. AZOIC DIAZO COMPONENT 6
SY - C.I. 37025
SY - CCRIS 2317
SY - DEVOL ORANGE B
SY - DEVOL ORANGE SALT B
SY - DIAZO FAST ORANGE GR
SY - FAST ORANGE BASE GR
SY - FAST ORANGE BASE JR
SY - FAST ORANGE GR BASE
SY - FAST ORANGE O BASE
SY - FAST ORANGE O SALT
SY - FAST ORANGE SALT GR
SY - FAST ORANGE SALT JR
SY - HILTONIL FAST ORANGE GR BASE
SY - HILTOSAL FAST ORANGE GR SALT
SY - HINDASOL ORANGE GR SALT
SY - HSDB 1132
SY - NATASOL FAST ORANGE GR SALT
SY - O-AMINONITROBENZENE
SY - o-NITRANILINE
SY - o-NITROANILINE
SY - ONA
SY - ORANGE BASE CIBA II
SY - ORANGE GRS SALT
SY - ORANGE SALT CIBA II
SY - ORANGE SALT IRGA II
SY - Orthonitroaniline
SY - 1-AMINO-2-NITROBENZENE

SY - 2-AMINONITROBENZENE
SY - 2-NITROANILINE
SY - 2-NITROBENZENAMINE

RDO -

o ORAL RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

o REVIEW DATES : 06/23/92

RDI - NO DATA
CAREV- NO DATA
CARO - NO DATA
CARI - NO DATA
CARDR- NO DATA

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU- NO DATA

WQCAQ- NO DATA

MCLG - NO DATA

MCL - NO DATA

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC - NO DATA

SARA - NO DATA

RCRA - NO DATA

TSCA - NO DATA

OREF - NO DATA

IREF - NO DATA

CREF - NO DATA

HAREF - NO DATA

Methoxychlor

1 - IRIS

NAME - Methoxychlor

RN - 72-43-5

IRSN - 368

DATE - 931201

UPDT - 12/01/93, 15 fields

STAT - Oral RfD Assessment (RDO) on-line 08/01/91

STAT - Inhalation RfC Assessment (RDI) message 12/01/93

STAT - Carcinogenicity Assessment (CAR) on-line 10/01/90

STAT - Drinking Water Health Advisories (DWHA) withdrawn 12/01/93

STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92

IRH - 09/07/88 CAR Carcinogen summary on-line

IRH - 02/01/89 CARDR Primary contact's phone number corrected

IRH - 06/01/89 CARDR Secondary contact deleted

IRH - 12/01/89 REFS Bibliography on-line

IRH - 05/01/90 RDO Oral RfD now under review

IRH - 09/01/90 RDO Oral RfD summary on-line

IRH - 09/01/90 HADV Health Advisory on-line

IRH - 09/01/90 OREF Oral RfD references added

IRH - 09/01/90 HAREF Health Advisory references added

IRH - 10/01/90 CAR Text edited

IRH - 08/01/91 RDO Khera citation year corrected

IRH - 08/01/91 OREF Khera reference year corrected

IRH - 12/01/91 RDI Inhalation RfC now under review

IRH - 01/01/92 EXSR Regulatory Action section on-line

IRH - 04/01/92 RDI Inhalation RfC message on-line

IRH - 04/01/92 IREF Inhalation RfC references added

IRH - 12/01/93 RDI Replaced with expanded assessment

IRH - 12/01/93 HADV Health Advisory withdrawn

IRH - 12/01/93 IREF References revised

IRH - 12/01/93 HAREF Health Advisory references withdrawn

RLEN - 57001

SY - 2,2-di-p-anisyl-1,1,1-trichloroethane

SY - DMDT

SY - marlate

SY - methoricide

SY - Methoxychlor

SY - methoxy-DDT

SY - moxie

SY - 1,1,1-trichloro-2,2-bis(p-methoxyphenyl)ethane

RDO -

o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Excessive loss of litters	NOEL: 5.01 mg/kg/day LEL: 35.5 mg/kg/day	1000 mg/kg/day	1	5E-3
Rabbit Teratology Study				

Kincaid Enterprises,
1986

*Conversion Factors: Actual dose tested

o ORAL RFD STUDIES :

Kincaid Enterprises, Inc. 1986. MRID No. 0015992. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Young adult female New Zealand White rabbits were randomized by a computerized process which assigned 17 animals each into 3 dose groups, 5.01, 35.5, and 251.0 mg/kg/day, and a control (a total of 68 animals). The females were artificially inseminated and the day of insemination considered as gestation day 0. All animals were dosed from days 7 through 19 of gestation. Animals were observed twice daily for mortality and moribundity, further they were observed once daily for clinical signs of toxicity. Individual body weights were taken on gestation days 0, 7, 10, 14, 20, 24, and 29. All surviving dams were sacrificed on gestation day 29.

Maternal toxicity was observed as excessive loss of litters (abortions) in the mid- and high-dose groups along with statistically significant decreases in body weight gain during the dosing period for both mid- and high-dose groups and in the mid dose following the dosing period and overall for the gestation period (the high dose was not analyzed due to total loss of litters). There also was an increase in clinical signs in both the mid- and high-dose groups; the deaths at the high dose were attributed to compound administration. The high incidence of lung agenesis noted in fetuses of all dose groups was unusual. No specific toxicity was noted in the low dose (5.01 mg/kg/day).

The tentative LEL for maternal toxicity is 35.5 mg/kg/day based on excessive loss of litters. The tentative NOEL for maternal toxicity is 5.01 mg/kg/day.

o ORAL RFD UNCERTAINTY :

UF -- An uncertainty factor of 100 was used to account for the inter- and intraspecies differences. An additional UF of 10 was used to account for the poor quality of the critical study and for the incompleteness of the data base on chronic toxicity.

o ORAL RFD MODIFYING FACTOR :

MF -- None

o ORAL RFD COMMENTS :

Methoxychlor is considered to have an estrogenic activity. Several recent papers in the open literature have addressed this action of methoxychlor. Kupfer and Bulger (1987) found that both methoxychlor and metabolites have estrogen-like activity with several metabolites having proestrogen activity. They used an in vitro system involving rat liver microsomes and NADPH for a metabolizing system with estrogen receptors from immature rat uteri as a detection system.

Gray et al. (1989) investigated the effects of methoxychlor on the pubertal development and reproductive function in the male and female rat (Long-Evans hooded) by dosing rats from gestation, weaning, lactation, through puberty with either 25, 50, 100, or 200 mg/kg/day of methoxychlor. In females they found an acceleration of vaginal opening, abnormal estrus cycle, inhibition of luteal function and a blockage of implantation. In males they found an inhibition of somatic growth and accessory gland weight, elevated pituitary and serum prolactin levels, and a suppression of testicular Leydig cell function. Some of these effects occurred at levels as low as 25 mg/kg/day. These observations are consistent with the earlier reports that Methoxychlor mimics estrogen both in vivo and in vitro.

Goldman et al. (1986) investigated the subchronic effects of methoxychlor on the rat (Long-Evans hooded) reproductive system by dosing for 8 weeks with 25 or 50 mg/kg of methoxychlor by oral gavage. No effect was observed on the pituitary weight, serum LH, FSH, or prolactin levels and the pituitary LH or FSH concentrations. Pituitary prolactin levels were increased at both levels.

There was an increase in GnRH levels in the mediobasal hypothalamus at the high-dose level. The authors determined that the reproductive effects of methoxychlor are mediated in part by an increase in prolactin release which in turn influences the hypothalamic levels of GnRH. This may be considered an early effect of methoxychlor on the rat reproductive system.

Cummings and Gray (1987) of the US EPA Health Effects Research Laboratory found that methoxychlor affects the decidual cell response of the rat uterus, suggesting a direct effect of the compound on the uterus with no effects on uterine weight, serum progesterone levels, or corpora lutea maintenance. Long-term exposure to methoxychlor reduced fertility and induced fetotoxicity.

The effects of reduced fertility and fetotoxicity were noted in a 3-generation reproduction study (see study #4). Although the available data for these 3 studies were limited, it is apparent that methoxychlor at 1000 ppm produced reproductive effects in the form of reduced fertility index, reduced litter size, and reduced viability index.

Khera et al. (1978) on the teratogenicity of methoxychlor found that treatment of pregnant rats with either technical grade or formulation of methoxychlor produced maternal toxicity in the form of reduced body weight gain at all doses tested (50 to 400 mg/kg/day). Developmental toxicity was noted as fetotoxicity at doses of 200 and 400 mg/kg/day and as a dose-related increase of wavy ribs at 100, 200, and 400 mg/kg/day.

A 2-year chronic rat study by du Pont de Nemours & Co. (1951) reported a systemic NOEL of 100 ppm (5 mg/kg/day); a 2-year chronic study by Hodge, et al. (1952) reported a systemic NOEL of 200 ppm (10 mg/kg/day). Although these studies are not definitive, they, along with the submitted studies from the registrant, support the NOEL of 5.01 mg/kg/day used for the calculation of the RfD for methoxychlor.

Data Considered for Establishing the RfD

1) Teratology - rabbit: Principal study - see previous description; core grade supplementary (Kincaid Enterprises, Inc., 1986)

2) Teratology - rat: Dietary levels tested: 0, 200, 500, and 1250 ppm (10, 25, and 62.5 mg/kg/day); Female ChR-CD albino rats (animals were received pregnant) were administered methoxychlor in the diet on gestation days 6 through 15. There was maternal toxicity in the mid- and high-dose groups in the form of reduced body weight gain, reduced food consumption, increased postimplantation loss, and a decreased number of liver fetuses per dam. There was 1 and 2 dams in the mid- and high-dose groups, respectively, with total resorptions of litters. The increase in postimplantation loss resulted in a decrease in the litter size in the mid-and high-dose groups. There was an indication of 4 runts in one litter in the mid dose group, however, there was no change in the mean fetal weight among dose groups. The mid- and high-dose group had statistically significantly increased numbers of litters with wavy ribs. Study deficiencies included the following: no individual animal data were provided; animals were received pregnant; and although dosing was by feed, the concentration analysis of the diet, diet preparation schedule, and stability of the test compound in the diet mixtures was not provided. Therefore the tentative LEL is 500 ppm (25 mg/kg/day) based on the above effects. The tentative NOEL is 200 ppm (10 mg/kg/day).; core grade supplementary (E.I. du Pont de Nemours and Co., Inc., 1976a)

3) Teratology - rat: Dietary levels tested: 0, 34.6, 138.4, 242.2, and 346.0 mg/kg/day; Female Sprague-Dawley rats were dosed by gavage from gestation day 6 through 15. Control animals received corn oil in equivalent volumes to the test material which was administered at the high dose. There was evidence of reduced body weight gain at all doses tested. Further, at 138.4 mg/kg/day and above there was an increased number of resorptions, dead fetuses, and increased postimplantation loss. There was evidence of altered growth in the form of delayed ossification of skull bones and sternbrae and the reduced fetal body weight at the high dose. All doses tested had an increased incidence of hydronephrosis, and reduced or no ossification of skull bones, sternbrae and vertebrae along with wavy ribs. Study deficiencies include lack of stability and concentration analysis, dosing data, summary litter incidence, and maternal examination data. Based on the above effects observed at the lowest dose tested, the tentative LEL for maternal and developmental toxicity is 34.6 mg/kg/day. An NOEL for maternal and developmental toxicity could not be established.; core grade supplementary (Chemical Formulators, Inc. 1976b)

4) 3-Generation Reproduction - rat: Dietary levels tested: 0, 200, and 1000 ppm (0, 10, and 50 mg/kg/day); Male and female ChR-CD rats were administered methoxychlor in the diet for three generations. Three separate studies were conducted and reported in this study. The first reproduction study used dose levels of 0 and 200 ppm and the second reproduction study used dose levels of 0, 0 (2 control groups) and 1000 ppm. The third study was a pair feeding study with rats given 1000 ppm. The available data was limited for these 3 studies, however, it is apparent that methoxychlor at 1000 ppm produced reproductive effects in the form of reduced fertility index, reduced litter size, and reduced viability index. There was evidence of possible systemic toxicity at the 200 ppm dose, however, there was also evidence of reduced food consumption. Therefore the tentative NOEL and LEL are 200 ppm (10 mg/kg/day) and 1000 (50 mg/kg/day), respectively.; core grade supplementary (E.I. du Pont de Nemours and Co., Inc., 1966)

Other Data Reviewed:

1) Carcinogenicity Study - rat: Dietary levels tested: Male: 0, 360, 500, 720, and 1000 ppm (0, 18, 25, 36, and 50 mg/kg/day); Female: 0, 750, and 1500 ppm (0, 37.5, and 75 mg/kg/day); Male and female Osborne-Mendel rats were administered methoxychlor in the diet for 2 years. The initial dose levels for males were 360 and 720 ppm but were increased to 500 and 1000 ppm after week 30. Based on the data provided in this study, there is no substantial evidence that the MTD had been reached. The reduced male and female body weights noted in treated groups may be due to reduced food consumption (no food consumption data provided), also other studies with methoxychlor indicate that mixing the compound in the food tends to reduce food consumption and therefore weight.; core grade supplementary (U.S. Department of Health, Education, and Welfare, 1977a)

2) Carcinogenicity Study - mouse: Dietary levels tested: Male: 0, 1400, 1750, 2800, and 3500 ppm (0, 210, 262.5, 420, and 525 mg/kg/day); Female: 0, 750, 1000, 1500, and 2000 ppm (0, 112.5, 150, 225, and 300 mg/kg/day); Male and female B6C3F1 were administered methoxychlor in the diet for 78 weeks. The initial dose levels for males were 1400 and 2800 ppm while females initially received 750 and 1500 ppm. After week two, doses were increased to 1750 and 3500 ppm for males and to 1000 and 2000 ppm for females. Based on the data provided in this study, there is no substantial evidence that the MTD had been reached. The reduced body weights noted in treated males (high dose only) and in treated females (all dose levels) may be due to reduced food consumption (no food consumption data provided). Other studies with methoxychlor indicate that mixing the compound in the food tends to reduced food consumption and therefore weight.; core grade supplementary (U.S. Department of Health, Education, and Welfare, 1977b)

Data Gap(s): Chronic Rat Feeding/Carcinogenicity Study; Chronic Dog Feeding Study; Rat Reproduction Study; Rat Developmental toxicity Study; Rabbit Developmental toxicity Study; Chronic Mouse Feeding/Carcinogenicity Study

o ORAL RFD CONFIDENCE :

Study – Low
Data Base – Low
RfD – Low

The critical study is given a low confidence rating since no conclusions could be made relative to the maternal or developmental toxicity of Methoxychlor due to the total loss of litters in the high-dose group and the small number of litters available for evaluation in the mid-dose group. The data base is given a low confidence rating because of the lack definitive chronic toxicity studies. Low confidence in the RfD follows.

o ORAL RFD SOURCE DOCUMENT :

Source Document – This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation – Pesticide Registration Standard, August 1988; Pesticide Registration Files

o REVIEW DATES : 04/18/90, 05/17/90, 06/21/90
o VERIFICATION DATE : 06/21/90
o EPA CONTACTS :

George Ghali / OPP -- (703)557-7490

William Burnam / OPP -- (703)557-7491

RDI -

o INHALATION RFD SUMMARY :

The health effects data for methoxychlor were reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for the derivation of an inhalation RfC. The verification status for this chemical is currently NOT VERIFIABLE. For additional information on the health effects of this chemical, interested parties are referred to the U.S. EPA documentation listed below.

NOT VERIFIABLE status indicates that the U.S. EPA RfD/RfC Work Group deemed the database at the time of review to be insufficient to derive an inhalation RfC according to the Interim Methods for Development of Inhalation Reference Concentrations (U.S. EPA, 1990). This status does not preclude the use of information in cited references for assessment by others.

Derivation of an inhalation RfC for methoxychlor is not recommended at this time. No adequate long-term studies examining the effects of inhalation exposure to methoxychlor exist. No inhalation pharmacokinetic data exist for this compound. No data exist to definitively rule out portal-of-entry effects. The requirements for a minimal database have not been met (U.S. EPA, 1990).

Methoxychlor [2,2-bis(4-methoxyphenyl)-1,1,1-trichloroethane], also known as methoxy-DDT, is a pale yellow, crystalline organochlorine insecticide. It is used principally as a larvicide. Vapor pressure data on methoxychlor are not available. Methoxychlor is the p-methoxy derivative of the insecticide dichlorodiphenyltrichloroethane (DDT). Technical grade methoxychlor contains approximately 88% methoxychlor, with the remaining 12% comprising at least 50 impurities.

The only study of methoxychlor using inhalation exposure is that of Haag et al. (1950) who exposed two dogs, two rabbits, and 10 rats to an atmosphere containing micronized dust in which 10% recrystallized methoxychlor was mixed with Pyrax (composition not described) plus 3% Santo-Cel (a dehydrated silica gel) for 2 hours/day, 5 days/week. Concentrations and duration of exposures for three replicate experiments were reported as 300 mg/cu.m for 4 weeks, 360 mg/cu.m for 4 weeks, and 430 mg/cu.m for 5 weeks. This diluent was itself toxic and caused death and weight changes in the control dogs and rats at about the same incidence as the group exposed to methoxychlor. Further, it is not clear from the report whether the amount of diluent was normalized for all the exposed groups. The toxicity of DDT was also investigated in this study.

DDT and methoxychlor were of comparable toxicity in dogs and rabbits, but methoxychlor was less toxic than DDT in rats.

No reliable information is available on the effects of methoxychlor in humans, via inhalation or oral exposure. Ziem (1982) reported the case of a 49-year-old man who suffered from fatigue and bruising several weeks after he used a tomato dust pesticide containing methoxychlor. Two months after exposure he was diagnosed with aplastic anemia, and he died within 6 months. The man was well and had not been taking any drugs prior to exposure to methoxychlor. This is the only case of aplastic anemia reported in association with exposure to methoxychlor. Lehman (1949, as cited in U.S. EPA, 1987a) estimated that the lethal oral dose of methoxychlor in humans is 450 g (6.4 g/kg for a 70 kg human). Stein (1970) reported the results of an experiment in which 16 human volunteers (prisoners) were orally administered either 0.5, 1.0, or 2.0 mg/kg methoxychlor for 5 to 8 weeks.

Histopathological examination of biopsies of several tissues (liver, fat, bone marrow, and testicle) evidenced no abnormality. No weight disturbances or changes in clinical pathology (parameters measured not specified) were noted in the treated volunteers. Stein (1970) also reported the results of a study in which Sprague-Dawley rats (number not indicated) and Rhesus monkeys (3/group, sex not indicated) were administered 400-2500 mg/kg methoxychlor in 1% gum tragacanth by gavage for approximately 3 months (rats) or 6 months (monkeys). The rats demonstrated a dose-related depression in body weight gain after 4-6 weeks of treatment, but no weight disturbances were observed in the monkeys. No treatment-related effects on any of the clinical chemistry parameters measured were noted in either the rats or the monkeys. Similarly, no gross or microscopic evidence of treatment-related pathology was noted in either the rats or the monkeys. A decrease in hepatic triglycerides in both rats and monkeys was noted.

Several investigators have demonstrated that methoxychlor and its metabolites possess estrogenic properties (Bulger et al., 1978; Kupfer and Bulger, 1987). These estrogenic effects are manifested by changes in both male and female reproductive function and morphology in rodents. Administration of methoxychlor at rather high doses by gavage, in feed, or parenterally has been reported to stimulate the development of the reproductive tract in neonatal female rodents and their offspring, as evidenced by early vaginal opening, vaginal cornification, and an increase in the weight of reproductive organs (i.e., ovary and uterus) (Bulger et al., 1978; Eroschenko and Cooke, 1990; Gray et al., 1989; Harris et al., 1974). Methoxychlor administered to mature female rodents has been reported to inhibit reproductive function, as evidenced by inhibited folliculogenesis and atresia of follicles (Bal, 1984); decreased fertility; reduced implantations; and abnormal estrous cyclicity and/or persistent vaginal estrus (Gray et al., 1988, 1989; Martinez and Swartz, 1991). Atypical cell growth has also been noted in the uterus and oviducts (Eroschenko and Cooke, 1990; Gray et al., 1988). In a series of experiments conducted by Cummings and coworkers, it was demonstrated that the estrogenic, antifertility effects of methoxychlor are mediated in part by a direct effect on the uterus to suppress decidualization (Cummings and Gray, 1987), by suppression of serum progesterone levels (Cummings and Gray, 1989), and by accelerated transport of fertilized ova through the oviducts resulting in a loss of viable embryos that could account for the increase in preimplantation loss observed with methoxychlor (Cummings and Perreault, 1990). The estrogenic effects of methoxychlor have also been

observed with regard to behavior. Behaviors thought to be mediated by estrogen (running wheel activity and sexual behavior) were enhanced in intact and ovariectomized female rats treated with methoxychlor, and the enhanced behaviors were suppressed by progesterone, which is known to block the effects of estrogen (Gray et al., 1988).

Effects on male reproductive function have also been reported following the administration of methoxychlor to rodents. Bal (1984) reported inhibited spermatogenesis, degeneration of spermatogonia and spermatocytes, and cytoplasmic vacuolation in the epithelium of the ductus epididymis in male rats following the administration of 100-200 mg/kg/day methoxychlor. A decrease in seminal vesicle and caudal epididymal weight and caudal sperm count as well as delayed puberty were observed in neonatal rats administered 25-200 mg/kg/day methoxychlor for one generation, indicating that the endocrine function of the testes and pituitary gland were affected (Gray et al., 1989). Cooke and Eroschenko (1990) also noted that the development of the neonatal male rat reproductive tract was inhibited by methoxychlor administration, as evidenced by a decrease in serum testosterone levels and decreased DNA content of the seminal vesicles, bulbourethral glands, and the ventral prostate. Rats fed 2000 ppm methoxychlor for 90 days exhibited decreased prostate size and cell content (Shain et al., 1977). Goldman et al. (1986) hypothesized that part of methoxychlor's effects on male reproductive function may be mediated by a prolactinemic effect since administration of 25 or 50 mg/kg/day methoxychlor to 21-day-old male rats caused an increase in serum prolactin levels and an increase in hypothalamic-gonadotropin-releasing hormone levels.

Methoxychlor has been demonstrated to be fetotoxic. Khara et al. (1978) studied the effects of oral administration of 50, 100, 200, or 400 mg/kg/day methoxychlor to pregnant Wistar rats on gestational days 6-15. Two formulations of methoxychlor were used: (1) technical grade and (2) a formulation that was 50% methoxychlor (the composition of the remaining 50% was unknown). Maternal body weight gain was depressed in all treatment groups and remained depressed after removal of the uterine contents, implying an adverse effect on the dam. Treatment with either formulation of methoxychlor at the two highest doses resulted in a reduced number of rats with live fetuses at term and a reduced number of live fetuses per pregnancy. Reduced fetal weight gain was observed at the two highest dose levels with both formulations. An increased incidence of fetal skeletal anomalies (mostly wavy ribs) was observed at the two highest dose levels with both formulations.

Several chronic oral carcinogenicity bioassays have been conducted with methoxychlor (see review by Reuber, 1980), the results of which have been equivocal such that methoxychlor has yet to be classified as a carcinogen by the U.S. EPA. Aside from a depression in body weight gain observed in both rats fed at 1500 ppm and mice fed at 1994 ppm in a 2-year study conducted by NCI, no dose-related nonneoplastic effects were reported in these studies. Deichmann et al. (1967), however, fed 1000 ppm methoxychlor for 27 months to Osborne-Mendel rats and reported other nonneoplastic hepatic effects, including decreased absolute weight accompanied by hydropic swelling and some necrosis and congestion. Reuber (1980) reevaluated the slides from the carcinogenicity study of miniature swine and described the occurrence of moderate hyperplasia of the mammary gland with milk-like secretion, hyperplasia of the uterus, and chronic interstitial renal fibrosis. These

lesions are similar to those observed in the subchronic study in swine reported by Stein (1970) and Tegeris et al. (1966) and may be interpreted to be due to the estrogenic properties of methoxychlor.

A series of studies conducted in dogs and swine indicates that the two species respond differently with respect to the toxicity of methoxychlor (Stein, 1970; Tegeris et al., 1966). Technical grade methoxychlor (1, 2, or 4 g/kg) was administered in the feed 7 days/week to groups of six animals each (with 12 animals serving as controls) for up to 6 months. Clinical examinations were conducted daily, weights were recorded weekly, and blood samples were taken for hematological and clinical chemical analyses at 6-week intervals throughout the experiment. Bone marrow morphology and complete necropsies, with histopathological evaluation of approximately 18 tissues, were conducted at study termination. All dogs that were fed methoxychlor lost weight throughout the experiment, but, after an initial 8-week weight loss, the swine receiving the two lower doses of methoxychlor began to gain weight, whereas the high-dose swine continued to lose weight. Most of the medium-dose (5/6) and all of the high-dose dogs (6/6) began exhibiting clinical signs of toxicity after 6 weeks of treatment. Symptoms included nervousness and apprehension, progressing to salivation, fasciculations, tremors, hyperesthesia, mydriasis, tonic seizures, and tetanic convulsions. Most of these dogs died 3 weeks thereafter. The swine exhibited no clinical signs of toxicity. No treatment-related changes in any of the hematological parameters studied were noted in either the dogs or the swine. The dogs exhibited dose-dependent elevations in SGOT, SGPT, and alkaline phosphatase (AP). At 24-weeks exposure, the enzyme values of the high-exposure group relative to control values were increased eightfold for SGOT, 30-fold for SGPT, and 30-fold for AP, whereas the swine exhibited only a two-fold increase in BUN. The only changes attributed to methoxychlor noted at gross and microscopic examination in dogs (including the liver) were a dose-dependent absence of adipose tissue from the normal depots and congestion of the small intestinal mucosa (without accompanying histopathology). In the swine, advanced chronic renal nephritis, hyperplastic and hypertrophic mammary glands, and hypertrophic uteri were noted in the treated animals. These latter effects on sex organs are most likely due to the estrogenic properties of methoxychlor.

Very little quantitative information is available on the toxicokinetics of methoxychlor, and the available information is for oral or parenteral routes of exposure only. Absorption of methoxychlor from the gastrointestinal tract can be inferred from the observation of toxic effects following oral administration. Kapoor et al. (1970) administered radiolabeled methoxychlor to mice and found that 98.3% of the administered radioactivity was eliminated within 24 hours, mostly in the feces. A number of studies show that methoxychlor does not accumulate in the body to any appreciable degree (e.g., Villeneuve et al., 1972), but accumulation of methoxychlor in fat has been observed following administration of very high dietary levels of methoxychlor (U.S. EPA, 1987b). Methoxychlor is metabolized in the liver to readily excretable polar compounds (U.S. EPA, 1987b). Methoxychlor and 26 metabolites were identified in the feces, urine, and bile of intact, colostomized, and bile-fistulated chickens orally administered methoxychlor (Davison et al., 1984). Lactating goats also eliminate methoxychlor and its metabolites primarily in the feces (Davison et al., 1982). The results of studies by Villeneuve et al. (1972) indicate that methoxychlor does not induce hepatic microsomal enzymes.

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o REVIEW DATES : 11/07/91

CAREV-

o CLASSIFICATION : D; not classified as to human carcinogenicity

o BASIS FOR CLASSIFICATION : Human data are unavailable, and animal
evidence is inconclusive.

o HUMAN CARCINOGENICITY DATA :

None.

o ANIMAL CARCINOGENICITY DATA :

A number of chronic dietary studies have been done to test the carcinogenicity of methoxychlor in rats and mice (Nelson and Fitzhugh, 1951; Hodge et al., 1952, 1966; Radomski et al., 1965; Davis, 1969; Deichmann et al., 1967; NCI, 1978). In addition, two limited studies using mice (Hodge et al., 1966) have been performed by subcutaneous administration and skin application. Reuber (1980) reviewed these chronic studies, reevaluating raw data and the histological sections when possible.

In the Nelson and Fitzhugh (1951) study, Osborne-Mendel rats (12 rats/sex/group) ingested 0, 10, 25, 100, 200, 500 or 2000 ppm methoxychlor in the diet for 2 years. Animals were examined for gross lesions. Histological preparations were made only from the gross lesions found at autopsy. In the highest dose group four hepatic cell adenomas were observed, but this was not a statistically significant increase. No other changes or malignant lesions were noted in other organs. In his review of this study, Reuber (1980) concluded that the incidence of hepatic neoplasms in the treated animals was significantly greater than that in controls when hyperplastic nodules were included.

Groups of 25 male and 25 female rats (strain not specified) ingested 0, 25, 200, or 1600 ppm methoxychlor in the diet for 2 years (Hodge et al., 1952). At the end of 2 years, surviving animals were killed and many organs were examined grossly and histopathologically. In treated female rats, a greater number of total tumors was observed compared with controls. The authors considered this increase to be of no biological relevance because there was no significant increase in tumors of any one organ. Interpretation of these results is limited by the fact that many of the animals were not accounted for at the end of the study and that the liver was not routinely examined histologically.

Radomski et al. (1965) administered methoxychlor for 2 years in the diet at levels of 0 and 80 ppm to Osborne-Mendel rats (30 rats/sex/group). No increase in tumor incidence was found in the treated rats as compared with controls. Methoxychlor was also administered under the same regimen in a mixture with aramite, DDT, and thiourea at concentrations of 50 ppm each to 50 rats/sex/group. In this study an apparent increase in total tumors was observed in animals treated with the mixture as compared to controls.

Deichmann et al. (1967) administered methoxychlor in the diet to Osborne-Mendel rats (30/sex/dose) at levels of 0 and 1000 ppm for 27 months. The

concentration was chosen to be 50% of the highest dose reported in the Nelson and Fitzhugh study (1951). An increase in the number of total tumors was observed in treated males as compared with controls, but the increase was not statistically significant.

NCI (1978) tested groups of 50 male and 50 female Osborne-Mendel rats and 50 male and 50 female B6C3F1 mice. Control groups of each species consisted of 20 males and 20 females. Rats were exposed to technical grade methoxychlor (95% pure) in the diet for 78 weeks, followed by a 33-week observation period without exposure to the test compound. Concentrations given the low-dose male rats were 360 ppm for the first 29 weeks followed by 500 ppm for the next 49 weeks. The high-dose group was given 720 ppm for 29 weeks, 1000 ppm for the following 29 weeks, then 1000 ppm administered in a cyclic pattern for 20 weeks of one dosage-free week followed by 4 weeks of treatment. The low-dose female rats were given 750 ppm for the entire 78 weeks. The high-dose group received 1500 ppm for 55 weeks followed by 23 weeks of the cyclic pattern of administration at the same concentration. The time-weighted average (TWA) concentration for the high- and low-dose groups, respectively, was 845 and 448 mg/kg for the male rats and 1385 and 750 mg/kg for female rats, respectively.

Male mice were given a concentration of 1400 ppm for 1 week, then 1750 ppm for 77 weeks or 2800 ppm for 1 week, then 3500 ppm for 77 weeks. Female mice were given concentrations of 750 ppm for 1 week, then 1000 ppm for 77 weeks or 1500 ppm for 1 week, then 2000 ppm for 77 weeks. The mice were observed for an additional 15 weeks with no methoxychlor treatment. The TWA concentration for high- and low-dose groups, respectively, was 3491 and 1746 mg/kg for the male mice and 1994 and 997 mg/kg for female mice. Necropsy was performed on all animals that died spontaneously or were killed when moribund or at the termination of the study. Histological examinations were performed on major organs and on any gross lesions of all animals, except where cannibalism or autolysis precluded such studies.

The only tumors observed at a higher incidence than in controls were hemangiosarcomas in male rats (1/20 control, 9/50 low-, 2/50 high-dose groups). Although historically this tumor type is not frequently observed in this strain or rats, the authors concluded that the increase was not a good indicator of the carcinogenicity of methoxychlor because the response was neither dose-related nor statistically different from control values. Other tumors observed in the treated rats also occurred in the controls at the same frequency. NCI concluded that under this experimental regimen, methoxychlor was not tumorigenic to Osborne-Mendel rats. In mice, a variety of tumors was observed, but the incidence was similar in both control and experimental groups. Recent reviews by Greiesemer and Cueto (1980) and Harper et al. (1982) indicated that the bioassays did not meet the current criteria for maximum tolerated doses and so were not powerful enough to detect carcinogenicity. The evidence of carcinogenicity was, therefore, judged to be inconclusive, rather than negative.

In the Davis (1969) study, male and female BALB/c and C3H mice (100/sex/strain) were fed diets containing 0 or 750 ppm methoxychlor for 2 years. Liver tumors were found in male and female BALB/c mice and in male C3H mice. Carcinomas of the testes were observed in male BALB/c mice. It was the author's preliminary judgment that the data did not show that

methoxychlor was carcinogenic but suggested that a more complete statistical analysis was needed. In reviewing the original data, Reuber (1980) concluded that the increased incidences of liver carcinoma in C3H males and in BALB/c males and females were statistically significant, as well as increases in testicular carcinoma in BALB/c males and neoplasms at all sites in male and female BALB/c mice.

Nelson and Radomski (1953) fed methoxychlor at a dose of 300 mg/kg/day to four dogs. Two of the dogs died early in the study, but two female dogs survived the dosing period of 3.5 years. Liver foci were observed in one dog, and the other was described as exhibiting slight fibrosis in the liver. Reuber (1980) reexamined the histological sections and reported that one dog had developed liver carcinoma. The small number of animals used in this study precludes any definitive interpretation of these findings.

There is considerable disagreement between Reuber and the original authors in the interpretation of the histology and data from several of the chronic studies. NCI (1978), IARC (1979), and U.S. EPA (1983) have concluded that the experimental evidence does not support the contention that methoxychlor is a carcinogen. U.S. EPA (1987) has suggested that the differences in the conclusions may be due in part to the difficulty in distinguishing between regenerative hyperplasia, hyperplastic nodules, benign neoplasia, and malignant neoplasia, as well as the use of inappropriate control data in some of Reuber's statistical analyses.

o SUPPORTING DATA :

In mutagenicity assays, negative results were obtained (with or without metabolic activation) in bacteria, yeast, in assays of methoxychlor-induced DNA damage, or in assays of unscheduled DNA synthesis in mammalian cell cultures (Probst et al., 1981). A weakly positive increase was observed in a transformation study using BALB/3T3 cell line (Dunkel et al., 1981). Methoxychlor is a structural analog of DDT.

CARO - NO DATA

CARI - NO DATA

CARDR-

o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1987, 1983

The 1987 Drinking Water Criteria document received OHEA review. The Multimedia Risk Assessment received Agency review.

DOCUMENT

o REVIEW DATES : 10/07/87
o VERIFICATION DATE : 10/07/87
o EPA CONTACTS :

Dharm V. Singh / OHEA -- (202)260-5889

HAONE-

The Health Advisory for methoxychlor has been withdrawn on 12/01/93. A revised Health Advisory is in preparation by the Office of Water. For further details contact Amal Mahfouz / OST – (202)260-9568.

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 1.0E+2 ug/L

Fish Consumption Only: None

Considers technological or economic feasibility? – NO

Discussion – This value is the same as the drinking water standard and approximates a safe level assuming consumption of contaminated organisms and water.

Reference – Quality Criteria for Water, July 1976 (PB-263943).

EPA Contact – Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute – None

Chronic – 3.0E-2 ug/L

Marine:

Acute -- None

Chronic -- 3.0E-1 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference -- Quality Criteria for Water, July 1976 (PB-263943).

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0.04 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- A MCLG of 0.04 mg/L is promulgated based on potential adverse effects (developmental toxicity) reported in a rabbit study. The MCLG is based upon a DWEL of 2 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.04 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- EPA has promulgated a MCL equal to the MCLG of 0.04 mg/L.

Monitoring requirements -- All systems monitored initially for four consecutive quarters every three years; repeat monitoring dependent upon detection, vulnerability status and size.

Analytical methodology -- Microextraction/gas chromatography (EPA 505); gas chromatography/electron capture detector (EPA 508); liquid-solid extraction and column gas chromatography/mass spectrometry (EPA 525).
PQL=0.001 mg/L.

Best available technology -- Granular activated carbon

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___ IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___ IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

SMCL - NO DATA

FISTD-

Status -- Issued (1988)

Reference -- Methoxychlor Pesticide Registration Standard. December, 1988
(NTIS No. PB89-138523).

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

No data available

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ for methoxychlor is based on aquatic toxicity as established under CWA Section 311 (40 CFR 117.3). The available data indicate that the aquatic 96-Hour Median Threshold Limit is less than 0.1 ppm, which

corresponds to an RQ of 1 pound.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

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OREF - Cummings, A.M. and L.E. Gray, Jr. 1987. Methoxychlor affects the decidual cell response of the uterus but not other progestational parameters in female rats. Toxicol. Appl. Pharmacol. 90: 330-336.

OREF - E.I. du Pont de Nemours and Company, Inc. 1951. MRID No. 00029282. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

OREF - E.I. du Pont de Nemours and Company, Inc. 1966. MRID No. 00108732, 00113276. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

OREF - E.I. du Pont de Nemours and Company, Inc. 1976a. MRID No. 00062704. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

OREF - Goldman, J.M., R.L. Cooper, G.L. Rehnberg, J.F. Hein, W.K. McElroy and L.E. Gray, Jr. 1986. Effects of low subchronic doses of methoxychlor on the rat hypothalamic-pituitary reproductive axis. Toxicol. Appl. Pharmacol. 86: 474-483.

OREF - Gray, L.E., Jr., J. Ostby, J. Ferrell, et al. 1989. A dose-response analysis of methoxychlor-induced alterations of reproductive development and function in the rat. Fund. Appl. Toxicol. 12: 92-108.

OREF - Hodge, H.C., E.A. Maynard and H.J. Blanchet, Jr. 1952. Chronic oral toxicity tests of methoxychlor (2,2-Di-(P-methoxyphenyl)-1,1,1-trichloroethane) in rats and dogs. J. Pharmacol. Exp. Ther. 104: 60-66.

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45: 435-444.

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- OREF - Kupfer, D. and W.H. Bulger. 1987. Metabolic activation of pesticides with proestrogenic activity. *Fed. Proceed.* 48(5): 1864-1869.
- OREF - U.S. DHEW (U.S. Department of Health, Education, and Welfare). 1977a. MRID No. 00026602. Available from EPA. Write to FOI, EPA, Washington, DC 20460.
- OREF - U.S. DHEW (U.S. Department of Health, Education, and Welfare). 1977b. MRID No. 00026602. Available from EPA. Write to FOI, EPA, Washington, DC 20460.
- IREF - Bal, H.S. 1984. Effect of methoxychlor on reproductive systems of the rat (41861). *Proc. Soc. Exp. Biol. Med.* 176(2): 187-196.
- IREF - Bulger, W.H., R.M. Muccitelli, and K. Kupfer. 1978. Studies on the in vivo and in vitro estrogenic activities of methoxychlor and its metabolites. Role of hepatic mono-oxygenase in methoxychlor activation. *Biochem. Pharmacol.* 27(20): 2417-2423.
- IREF - Cooke, P.S. and V.P. Eroschenko. 1990. Inhibitory effects of technical grade methoxychlor on development of neonatal male mouse reproductive organs. *Biol. Reprod.* 42(3): 585-596.
- IREF - Cummings, A.M. and L.E. Gray. 1987. Methoxychlor affects the decidual cell response of the uterus but not other progestational parameters in female rats. *Toxicol. Appl. Pharmacol.* 90(2): 330-336.
- IREF - Cummings, A.M. and L.E. Gray. 1989. Antifertility effect of methoxychlor in female rats -- dose- and time-dependent blockade of pregnancy. *Toxicol. Appl. Pharmacol.* 97(3): 454-462.
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- IREF - Davison, K.L., C.H. Lamoureux, and V.J. Feil. 1984. Methoxychlor metabolism in chickens. *J. Agric. Food Chem.* 32(4): 900-908.
- IREF - Deichmann, W.B., M. Keplinger, F. Sala, and E. Glass. 1967. Synergism among oral carcinogens. IV. The simultaneous feeding of four tumorigens to rats. *Toxicol. Appl. Pharmacol.* 11(1): 88-103.
- IREF - Eroschenko, V.P. and P.S. Cooke. 1990. Morphological and biochemical alterations in reproductive tracts of neonatal female mice treated with the pesticide methoxychlor. *Biol. Reprod.* 42(3): 573-583.
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- IREF - Reuber, M.D. 1980. Carcinogenicity and toxicity of methoxychlor. *Environ. Health Perspect.* 36: 205-219.
- IREF - Shain, S.A., J.C. Shaeffer, and R.W. Boesel. 1977. The effect of chronic ingestion of selected pesticides upon rat ventral prostate homeostasis. *Toxicol. Appl. Pharmacol.* 40(1): 115-130.
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- CREF - Nelson, A.A. and O.G. Fitzhugh. 1951. Pathological changes produced in rats by feeding of methoxychlor at levels up to 0.2% of diet for 2 years. Prepared as a memorandum to A.J. Lehman, Food and Drug Administration, Washington, DC, June 9. (Cited in U.S. EPA, 1983)
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- CREF - U.S. EPA. 1983. Multimedia Risk Assessment for Methoxychlor. Environmental Criteria and Assessment Office, Office of Water Regulation and Standards, Cincinnati, OH. (Draft: August, 1983).
- CREF - U.S. EPA. 1987. Drinking Water Criteria Document for Methoxychlor. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.
- HAREF- Not available at this time

[IRIS] SS 2 /cf?
USER:

Aldrin

1 - IRIS
 NAME - Aldrin
 RN - 309-00-2
 IRSN - 127
 DATE - 930701
 UPDT - 07/01/93, 3 fields
 STAT - Oral RfD Assessment (RDO) on-line 03/01/88
 STAT - Inhalation RfC Assessment (RDI) no data
 STAT - Carcinogenicity Assessment (CAR) on-line 07/01/93
 STAT - Drinking Water Health Advisories (DWHA) no data
 STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
 IRH - 09/30/87 CAR Carcinogenicity section added
 IRH - 03/01/88 CARO Confidence statement revised
 IRH - 12/01/88 CARO Corrected slope factor in text
 IRH - 09/01/89 CAREV Ditraglia reference changed to Ditraglia et al.
 IRH - 09/01/89 CAREV Deichmann reference changed to Deichmann et al.
 IRH - 09/01/89 CARO Body weight for mice corrected to kg
 IRH - 09/01/89 REFS Bibliography on-line
 IRH - 01/01/91 CAR Text edited
 IRH - 01/01/91 CAR Inhalation slope factor removed (global change)
 IRH - 01/01/92 RDO Secondary contact changed
 IRH - 01/01/92 EXSR Regulatory actions updated
 IRH - 07/01/93 CARDR Secondary contact's phone number changed
 RLEN - 22408
 SY - Aldrex
 SY - Aldrin
 SY - Aldrite
 SY - Aldrosol
 SY - 1,4:5,8-Dimethanonaphthalene, 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-
 SY - Hexahydro-, (1 alpha, 4 alpha, 4a beta, 5 alpha, 8 alpha, 8a beta)-
 SY - 1,4:5,8-Dimethanonaphthalene,
 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-
 SY - Drinox
 SY - ENT 15,949
 SY - 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-1,4,5,8-Dimethanonapht
 halene
 SY - 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-1,4-endo-exo-5,8-
 SY - Dimethanonaphthalene
 SY - 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-exo-1,4-endo-5,8-
 SY - Dimethanonaphthalene
 SY - Hexachlorohexahydro-endo-exo-Dimethanonaphthalene
 SY - HHDN
 SY - NCI-C00044
 SY - Octalene
 SY - Seedrin

RDO -

o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver toxicity	NOAEL: none	1000	1	3E-5
		mg/kg/day		
Rat Chronic Feeding	LOAEL: 0.5 ppm diet			

Study (0.025 mg/kg/day)

Fitzhugh et al., 1964

*Conversion Factors: 1 ppm = 0.05 mg/kg/day (assumed rat food consumption)

o ORAL RFD STUDIES :

Fitzhugh, O.G., A.A. Nelson, and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.

Groups of 24 rats (12/sex) were fed aldrin in the diet at levels of 0, 0.5, 2, 10, 50, 100, or 150 ppm for 2 years. Liver lesions characteristic of chlorinated insecticide poisoning were observed at dose levels of 0.5 ppm and greater. These lesions were characterized by enlarged centrilobular hepatic cells, with increased cytoplasmic oxyphilia, and peripheral migration of basophilic granules. A statistically significant increase in liver-to-body weight ratio was observed at all dose levels. Kidney lesions occurred at the highest dose levels. Survival was markedly decreased at dose levels of 50 ppm and greater.

Additional data are fairly supportive. Effect and no-effect levels are similar (to those found for rats) for liver effects in dogs after 15 months' exposure to aldrin in the diet. Liver effects were observed at slightly higher doses in several other subchronic-to-chronic rat and dog studies. Short-term exposure to higher doses resulted in mortality for a number of species.

o ORAL RFD UNCERTAINTY :

UF -- The composite UF of 1000 encompasses the uncertainty of extrapolation from animals to humans, the uncertainty in the range of human sensitivities, and an additional uncertainty because the RfD is based on a LOAEL rather than a NOAEL.

o ORAL RFD MODIFYING FACTOR :

MF -- None

o ORAL RFD COMMENTS :

None.

o ORAL RFD CONFIDENCE :

Study -- Medium
Data Base -- Medium
RfD -- Medium

The principal study, designed as a carcinogenesis bioassay, is strong in histopathologic analysis but lacks other toxicologic parameters, and is therefore rated medium. The data base is fairly extensive, and generally supportive, but is rated medium because of the lack of NOELs for some studies.

Also, no chronic data exist for the dog, which may be a more sensitive species than the rat. Medium confidence in the RfD follows.

o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1982. Toxicity-Based Protective Ambient Water Levels for Various Carcinogens. Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-431. Internal review draft.

The RfD has been reviewed internally by ECAO-Cin.

o REVIEW DATES : 12/18/85
o VERIFICATION DATE : 12/18/85
o EPA CONTACTS :

Michael L. Dourson / OHEA -- (513)569-7533

Moiz Mumtaz / OHEA -- (513)569-7553

RDI - NO DATA
CAREV-

o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Orally administered aldrin produced significant increases in tumor responses in three different strains of mice in both males and females. Tumor induction has been observed for structurally related chemicals, including dieldrin, a metabolite.

o HUMAN CARCINOGENICITY DATA :

Inadequate. Two studies of workers exposed to aldrin and dieldrin (a metabolite of aldrin) did not find these workers to have an excess risk of cancer. Both studies, however, were limited in their ability to detect an excess of deaths from cancer. Van Raalte (1977) observed two cases of cancer (gastric and lymphosarcoma) among 166 pesticide manufacturing workers exposed 4 to 19 years and followed from 15 to 20 years. Exposure was not quantified, and workers were also exposed to other organochlorine pesticides (endrin and telodrin). A small number of workers was studied, the mean age of the cohort (47.7 years) was low, the number of expected deaths was not calculated, and the duration of exposure and of latency was relatively short.

In a retrospective mortality study, Ditraglia et al. (1981) reported no increased incidence of deaths from cancer among 1155 organochlorine pesticide manufacturing workers (31 observed vs. 37.8 expected, SMR=82). This result was not statistically significant. Workers were employed for 6 or more months and followed for 13 or more years (24,939 person-years). Workers with no exposure (for example, office workers) were included in the cohort. Vital status was not known for 112 (10%) of the workers, and these workers were assumed to be alive; therefore, additional deaths may have occurred but were not observed. Exposure was not quantified and workers were also exposed to

other chemicals and pesticides (including endrin). An increased incidence of deaths from cancer was seen at several specific sites: esophagus (2 deaths observed, SMR=235), rectum (3, SMR=242); liver (2, SMR=225), and lymphatic and hematopoietic system (6, SMR=147); but these site-specific incidences were not statistically significant.

o ANIMAL CARCINOGENICITY DATA :

Sufficient. Davis and Fitzhugh (1962) fed a group of 215 male and female C3HeB/Fe mice a dietary mixture containing 10 ppm aldrin for up to 2 years. The control group consisted of 217 mice. The aldrin-treated mice died 2 months earlier than controls. Intercurrent disease, pneumonia, and intestinal parasitism may have influenced the long-term survival rate. A statistically significant increase of hepatomas was reported in the treated animals as compared with controls. An independent reevaluation of the liver lesions showed most of the hepatomas to be liver carcinomas (Epstein, 1975). In a follow-up study, Davis (1965) administered aldrin at 0 or 10 ppm in the diet to 100 male and 100 female C3H mice for 2 years. The incidence of hepatic hyperplasia and benign hepatomas in the aldrin group was approximately double that of controls, whereas the number of hepatic carcinomas was about the same.

Neither study provided a detailed pathologic examination or data separated by sex.

Aldrin (95% pure) was administered in the diet to 50 male and 50 female B6C3F1 mice at TWA doses of 4 and 8 ppm or 3 and 6 ppm. Treatment was for 80 weeks, and animals were observed for an additional 10 to 13 weeks (NCI, 1978). In male mice, there was a significant dose-related increase in hepatocellular carcinomas when compared with matched or pooled controls.

Treon and Cleveland (1955) administered aldrin in the diet to 40 Carworth rats/sex at concentrations of 2.5, 12.5, or 25 ppm for a period of 2 years. Forty animals/sex served as controls. Mortality of the treated rats was greater than controls, with 50% surviving in the 2.5 and 12.5 ppm groups and 40% surviving in the 25 ppm group at the end of the experiment. Cleveland (1966) reported that no apparent treatment-related tumors were present in the above study. Deichmann et al. (1970) fed 50 male and 50 female Osborne-Mendel rats aldrin (95% pure) at final concentrations of 20, 30, or 50 ppm for 31 months. Controls consisted of 100 rats/sex. There was no evidence of carcinogenic response in male or female rats fed aldrin. The NCI (1978) fed 50 Osborne-Mendel rats/sex aldrin (95% pure) at 30 or 60 ppm. Male rats were treated 111 to 113 weeks and followed for 37 to 38 weeks of observation, and female rats were treated for 80 weeks and followed for 32 to 33 weeks of observation. Aldrin produced no significant effect on the mortality of the rats of either sex. The tumors observed were randomly distributed, with no apparent relationship to aldrin treatment. Four additional bioassays observed no carcinogenic effect of aldrin in rats, but were considered inadequate for carcinogenicity assessment.

o SUPPORTING DATA :

Aldrin causes chromosomal aberrations in mouse, rat, and human cells (Georgian, 1974) and unscheduled DNA synthesis in rats (Probst et al., 1981) and humans (Rocchi et al., 1980) cells. Aldrin does not cause reverse

mutations in *S. typhimurium*, *E. coli*, or *S. marcesans*, or mitotic gene conversion in *S. cerevisiae* (Fahrig, 1974).

Five compounds structurally related to aldrin—dieldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid—have induced malignant liver tumors in mice. Chlorendic acid has also induced liver tumors in rats.

CARO -

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : Orally administered aldrin produced significant increases in tumor responses in three different strains of mice in both males and females. Tumor induction has been observed for structurally related chemicals, including dieldrin, a metabolite.
- o ORAL SLOPE FACTOR : $1.7E+1$ per (mg/kg)/day
- o DRINKING WATER UNIT RISK : $4.9E-4$ per (ug/L)
- o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
- o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-1 ug/L
E-5 (1 in 100,000)	2E-2 ug/L
E-6 (1 in 1,000,000)	2E-3 ug/L

o ORAL DOSE-RESPONSE DATA :

Tumor Type -- liver carcinoma

Test Animals -- mouse/C3H (Davis); mouse/B6C3F1, male (NCI)

Route -- diet

Reference -- Davis, 1965 (see table); NCI, 1978

Administered Dose (ppm)	Human Equivalent Dose (mg/kg-day)	Tumor Incidence	Reference
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females

0	0	2/53	Davis, 1965
10	0.104	72/85	reevaluated by Reuber
0	0	22/73	(cited in
10	0.104	75/91	Epstein, 1975)

0	0	3/20	NCI, 1978
4	0.04	16/49	
8	0.08	25/45	

o ADDITIONAL COMMENTS :

Body weights for mice were assumed to be 0.03 kg for purposes of dose conversion. The above data sets were used for calculation of the following slope factors: $2.3E+1$ per (mg/kg)/day for female C3H mice, $1.8E+1$ per (mg/kg)/day for male C3H mice, and $1.2E+1$ per (mg/kg)/day for male B6C3F1 mice. No strain or sex specificity was noted in the studies, since aldrin treatment induced liver tumors in all mouse strains tested. A geometric mean of $1.7E+1$ per (mg/kg)/day was thus chosen for the quantitative estimate, since all three slope factors were very similar.

The unit risk should not be used if the water concentration exceeds 20 ug/L, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

Adequate numbers of animals were treated for a large proportion of their lifetime. The route of treatment was appropriate. Slope factors calculated from three data sets from two independent assays were within a factor of 2. A slope factor for dieldrin, a major metabolite of aldrin, was determined to be $1.6E+1$, essentially identical to that of aldrin.

CARI -

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : Orally administered aldrin produced significant increases in tumor responses in three different strains of mice in both males and females. Tumor induction has been observed for structurally related chemicals, including dieldrin, a metabolite.
- o INHALATION UNIT RISK : $4.9E-3$ per (ug/cu.m)
- o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
- o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	$2E-2$ ug/cu.m
E-5 (1 in 100,000)	$2E-3$ ug/cu.m
E-6 (1 in 1,000,000)	$2E-4$ ug/cu.m

o INHALATION DOSE-RESPONSE DATA :

The unit risk was calculated from the oral data presented in CARO.

o ADDITIONAL COMMENTS :

The unit risk should not be used if the air concentration exceeds 2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

See CARO.

CARDR-

o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1986

The values in the 1986 Carcinogenicity Assessment for Aldrin/Dieldrin have been reviewed by the Carcinogen Assessment Group.

DOCUMENT

o REVIEW DATES : 03/22/87

o VERIFICATION DATE : 03/22/87

o EPA CONTACTS :

Dharm V. Singh / OHEA -- (202)260-5889

Jim Cogliano / OHEA -- (202)260-3814

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption -- $7.4E-5$ ug/L

Fish Consumption Only -- $7.9E-5$ ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria

represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 3.0E+0 ug/L
Chronic -- None

Marine:

Acute -- 1.3E+0 ug/L
Chronic -- None

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

No data available

MCL -

No data available

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (Final, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Microextraction/gas chromatography (EPA 505); electron-capture/gas chromatography (EPA 508); gas chromatographic/mass spectrometry (EPA 525).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

SMCL - NO DATA

FISTD-

Status -- Issued (1986)

Reference -- Aldrin Pesticide Registration Standard. December, 1986 (NTIS No. PB-87-183778).

EPA Contact -- Registration Branch / OPP (703)557-7760 / FTS 557-7760

FIREV-

Action -- Cancellations issued prior to RPAR/special review process (1974)

Considers technological or economic feasibility? -- YES

Summary of regulatory action -- All uses canceled except those in the following list: 1) subsurface ground insertion for termite control, 2) dipping of nonfood roots and tops, 3) moth-proofing by manufacturing processes in a closed system. Accelerated Decision of the Chief Administrative Law Judge (5/27/75) and the order Declining Review of the Accelerated Decision of the Administrative Law Judge issued by the Chief Judicial Officer (6/30/75); criterion of concern: carcinogenicity, bio-accumulation, wildlife hazard and other chronic effects.

Reference — 39 FR 37246 (10/18/74)

EPA Contact — Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) — 1 pound (Final, 1989)

Considers technological or economic feasibility? — NO

Discussion — The RQ for aldrin is 1 pound, based on its aquatic toxicity and its potential carcinogenicity. The available data, as established under the CWA Section 311 (40 CFR 117.3), indicate the aquatic 96-hour Median Threshold Limit for aldrin is less than 0.1 ppm. This corresponds to an RQ of 1 pound. In addition, aldrin has been identified as a potential carcinogen and assigned a hazard ranking of high, based on a potency factor of 180.00/mg/kg/day and weight-of-evidence group B2, which also corresponds to an RQ of 1 pound.

Reference — 54 FR 33418 (08/14/89)

EPA Contact — RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status — Listed

Reference — 52 FR 25942 (07/09/87)

EPA Contact — RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Fitzhugh, O.G., A.A. Nelson, and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.

- OREF - U.S. EPA. 1982. Toxicity-Based Protective Ambient Water Levels for Various Carcinogens. Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-431. Internal review draft.
- IREF - None
- CREF - Cleveland, F.P. 1966. A summary of work on aldrin and dieldrin toxicity at the Kettering Laboratory. Arch. Environ. Health. 13: 195.
- CREF - Davis, K.J. 1965. Pathology report on mice fed dieldrin, aldrin, heptachlor, or heptachlor epoxide for two years. Internal FDA memorandum to Dr. A.J. Lehrman, July 19.
- CREF - Davis, K.J. and O.G. Fitzhugh. 1962. Tumorigenic potential of aldrin and dieldrin for mice. Toxicol. Appl. Pharmacol. 4: 187-189.
- CREF - Deichmann, W.B., W.E. McDonald, E. Blum, et al. 1970. Tumorigenicity of aldrin, dieldrin and endrin in the albino rat. Ind. Med. 39(10): 426-434.
- CREF - Ditraglia, D., D.P. Brown, T. Namekata and N. Iverson. 1981. Mortality study of workers employed at organochlorine pesticide manufacturing plants. Scand. J. Environ. Health. 7(suppl 4): 140-146.
- CREF - Epstein, S.S. 1975. The carcinogenicity of dieldrin. Part 1. Sci. Total Environ. 4: 1-52.
- CREF - Fahrig, R. 1974. Comparative mutagenicity with pesticides. IARC Publ. (U.N.) 10: 161-181.
- CREF - Georgian, L. 1975. The comparative cytogenic effects of aldrin and phosphamidon. Mutat. Res. 31: 103-108.
- CREF - NCI (National Cancer Institute). 1978. Bioassays of aldrin and dieldrin for possible carcinogenicity. DHEW Publication No. (NIH) 78-821. NCI Carcinogenesis Tech. Rep. Ser. No. 21. NCI-C6-TR-21.
- CREF - Probst, G.S., R.E. McMahon, L.W. Hill, D.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 chemicals. Environ. Mutagen. 3: 11-32.
- CREF - Rocchi, P., P. Perocco, W. Alberghini, A. Fini and G. Prodi. 1980. Effect of pesticides on scheduled and unscheduled DNA synthesis of rat thymocytes and human lymphocytes. Arch. Toxicol. 45: 101-108.
- CREF - Treon, J.F. and F.P. Cleveland. 1955. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals, with special reference to aldrin and dieldrin. Agric. Food Chem. 3: 402-408.
- CREF - U.S. EPA. 1986. Carcinogenicity Assessment of Aldrin and Dieldrin. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC, for the Hazard Evaluation Division, Office of Pesticides and Toxic Substances, Office of Pesticide Programs, Washington, DC.
- CREF - Van Raalte, H.G.S. 1977. Human experience with dieldrin in perspective. Ecotoxicol. Environ. Safety. 1: 203-210.
- HAREF - None

[IRIS] SS 5 /cf?

USER:

Heptachlor epoxide

1 - IRIS

NAME - Heptachlor epoxide

RN - 1024-57-3

IRSN - 157

DATE - 930701

UPDT - 07/01/93, 4 fields

STAT - Oral RfD Assessment (RDO) on-line 03/01/91

STAT - Inhalation RfC Assessment (RDI) no data

STAT - Carcinogenicity Assessment (CAR) on-line 07/01/93

STAT - Drinking Water Health Advisories (DWHA) on-line 08/01/90

STAT - U.S. EPA Regulatory Actions (EXSR) on-line 04/01/93

IRH - 09/30/87 CAR Carcinogen summary on-line

IRH - 03/01/88 RDO Text clarified

IRH - 03/01/88 RDO Confidence levels revised

IRH - 03/01/88 CARO Confidence statement revised

IRH - 03/01/88 HADV Health Advisory on-line

IRH - 08/01/90 HADR Primary contact changed

IRH - 08/01/90 RCRA EPA contact changed

IRH - 01/01/91 CAR Text edited

IRH - 01/01/91 CARI Inhalation slope factor removed (global change)

IRH - 03/01/91 RDO Citations added

IRH - 03/01/91 REFS Bibliography on-line

IRH - 01/01/92 EXSR Regulatory actions updated

IRH - 04/01/92 CAREV Text revised

IRH - 04/01/93 WQCAQ Freshwater and marine values corrected

IRH - 07/01/93 CARDR Secondary contact's phone number changed

RLEN - 21668

SY - ENT 25,584

SY - EPOXYHEPTACHLOR

SY - HCE

SY - Heptachlor Epoxide

SY - 1,4,5,6,7,8,8-HEPTACHLORO-2,3-EPOXY-2,3,3a,4,7,7a-HEXAHYDRO-4,7-METHANO
INDENE

SY - 1,4,5,6,7,8,8-HEPTACHLORO-2,3-EPOXY-3a,4,7,7a-TETRAHYDRO-4,7-METHANOIND
AN

SY - 2,3,4,5,6,7,7-HEPTACHLORO-1a,1b,5,5a,6,6a-HEXAHYDRO-2,5-METHANO-2H-INDE
NO(1,2-

SY - b)OXIRENE

SY - HIPTACHLOR EPOXIDE

SY - 4,7-METHANOINDAN,

1,4,5,6,7,8,8-HEPTACHLORO-2,3-EPOXY-3a,4,7,7a-TETRAHYDRO-

SY - 2,5-METHANO-2H-OXIRENO(a)INDENE,

2,3,4,5,6,7,7-HEPTACHLORO-1a,1b,5,5a,6,6a-

SY - HEXAHYDRO-

SY - VELSICOL 53-CS-17

RDO -

o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
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Increased liver-to-body weight ratio in both males and females	NOEL: none LEL: 0.5 ppm (diet) (0.0125 mg/kg/day)	1000	1	1.3E-5
		mg/kg/day		

60-Week Dog Feeding Study

Dow Chemical Co.,
1958

*Conversion Factors: 1 ppm = 0.025 mg/kg/day (assumed dog food consumption)

o ORAL RFD STUDIES :

Dow Chemical Company. 1958. MRID No. 00061912. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Beagle dogs from 23 to 27 weeks of age were divided into five groups (3 females and 2 males) and given diets containing 0, 0.5, 2.5, 5 or 7.5 ppm of heptachlor epoxide for 60 weeks. Liver-to-body weight ratios were significantly increased in a treatment-related fashion. Effects were noted for both males and females at the LEL of 0.5 ppm. A NOEL was not established.

o ORAL RFD UNCERTAINTY :

UF – Based on a chronic exposure study, an uncertainty factor of 1000 was used to account for inter- and intraspecies differences and to account for the fact that a NOEL was not attained.

o ORAL RFD MODIFYING FACTOR :

MF – None

o ORAL RFD COMMENTS :

None.

Data Considered for Establishing the RfD:

1) 60-Week Feeding - dog: Principal study - see previous description; no core grade

2) 2-Generation Reproduction - dog: NOEL=1 ppm (0.025 mg/kg/day); LEL=3 ppm (0.075 mg/kg/day) (liver lesions in pups); Reproductive NOEL=5 ppm (0.125 mg/kg/day); Reproductive LEL=7 ppm (0.175 mg/kg/day) (pup survival); no core grade (Velsicol Chemical, 1973a)

3) 3-Generation Reproduction - rat: NOEL=5 ppm (0.25 mg/kg/day); LEL=10 ppm (0.5 mg/kg/day) (pup mortality); no core grade (Velsicol Chemical, 1959a)

4) 2-Year Feeding - rat: LEL=0.5 ppm (0.025 mg/kg/day) (LDT) (females - vacuolar changes in central hepatic lobule); NOEL not established; no core

grade (Velsicol Chemical, 1959b)

Other Data Reviewed:

1) Chronic Feeding Study - mouse: Heptachlor/Heptachlor Epoxide (1:3):
NOEL=none; LEL=1 ppm (LDT) (vacuolation, enlarged nucleus, hepatocytomegaly);
no core grade (Velsicol Chemical, 1973b)

2) Chronic Feeding Study - rat: Heptachlor/Heptachlor Epoxide (3:1):
NOEL=none; LEL=5 ppm (LDT) (liver-to-body weight increase in females); no core
grade (Velsicol Chemical, 1966)

3) 3-Generation Reproduction - rat: Heptachlor/Heptachlor Epoxide (3:1):
NOEL=7 ppm (HDT); LEL=none; no core grade (Velsicol Chemical, 1967)

Data Gap(s): Rat Teratology Study; Rabbit Teratology

o ORAL RFD CONFIDENCE :

Study -- Low
Data Base -- Medium
RfD -- Low

The principal study is of low quality and is given a low confidence rating.
Since the data base on chronic toxicity is complete but consists of low-
quality studies, the data base is given a medium to low confidence rating.
Low confidence in the RfD follows.

o ORAL RFD SOURCE DOCUMENT :

Pesticide Registration Standard, August 1986

o REVIEW DATES : 12/18/85, 09/16/86
o VERIFICATION DATE : 09/16/86
o EPA CONTACTS :

William Burnam / OPP -- (703)557-4791

George Ghali / OPP -- (703)557-7490

RDI - NO DATA

CAREV-

o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Sufficient evidence exists from rodent
studies in which liver carcinomas were
induced in two strains of mice of both sexes
and in CFN female rats. Several structurally
related compounds are liver carcinogens.

o HUMAN CARCINOGENICITY DATA :

Inadequate. There are no published epidemiologic evaluations of heptachlor epoxide. It is not commercially available in the United States, but is a product of heptachlor oxidation.

There were 11 case reports involving central nervous system effects, blood dyscrasias and neuroblastomas in children with pre-/postnatal exposure to chlordane and heptachlor (Infante et al., 1978). Since no other information was available, no conclusions can be drawn.

There were three epidemiologic studies of workers exposed to chlordane and/or heptachlor. One retrospective cohort study of pesticide applicators was considered inadequate in sample size and duration of follow-up. This study showed marginal statistically significant increased mortality from bladder cancer (3 observed) (Wang and McMahon, 1979a). Two other retrospective cohort studies were of pesticide manufacturing workers. Neither of them showed any statistically significant increased cancer mortality (Wang and McMahon, 1979b; Ditraglia et al., 1981). Both these populations also had confounding exposures from other chemicals.

o ANIMAL CARCINOGENICITY DATA :

Sufficient. Four long-term carcinogenesis bioassays of heptachlor epoxide have been reported. The major finding in mice has been an increased incidence of liver carcinomas. Davis (1965) fed groups of 100 male and 100 female C3H mice 0 or 10 ppm heptachlor epoxide for 2 years. Survival was generally low, with 50% of controls and 9.5% of treated mice living 2 years. A 2-fold increase in benign liver lesions (hepatic hyperplasia and benign tumors) over the controls was reported. Reevaluation by Reuber (1977b) revealed a significant increase in liver carcinomas in the dosed group (77/81 in females and 73/79 in males) over the controls (2/53 in females and 22/73 in males). The Velsicol Chemical Co. (1973) tested a 75:25 mixture of heptachlor epoxide:heptachlor in groups of 100 male and 100 female CD-1 mice. The mice were fed 0, 1, 5, and 10 ppm for 18 months. A statistically significant increase of hyperplasia was observed in the 5, and 10 ppm dose groups in both sexes; Reuber's reevaluation (U.S. EPA, 1985) resulted in a change in diagnosis for benign to liver carcinomas, thereby increasing the incidence of hepatic carcinomas ($p < 0.01$). Four independent pathologists concurred with Reuber's reevaluation.

The earliest bioassay with rats (Witherup et al., 1959) tested 25 male and 25 female CFN rats each at 0.5, 2.5, 5.0, 7.5, and 10 ppm for 108 weeks. The authors observed malignant and benign tumors randomly among test groups and controls. Reuber's reevaluation (1985) reported a significant increase of hepatic carcinomas above the controls at 5 and 10 ppm in the female rats. A reevaluation by Williams (1985) reported a significant increase of hepatic nodules at the 10 ppm level in the males over the controls. The Kettering Laboratory (Jolley et al., 1966) tested a mixture of 75:25 heptachlor:heptachlor epoxide in the diet of 25 female CD rats at 5, 7.5, 10, and 12.5 ppm for 2 years. Although no malignant lesions of the liver were observed, hepatocytomegaly was increased at 7.5, 10, and 12.5 ppm.

o SUPPORTING DATA :

Gene mutation assays indicate that heptachlor epoxide is not mutagenic in

bacteria (Moriya et al., 1983). In two mouse dominant lethal assays, heptachlor epoxide did not induce major chromosomal aberrations in male germinal cells (Arnold et al., 1977; Epstein et al., 1972). Ahmed et al. (1977) reported qualitative evidence of uncheduled DNA synthesis response in SV40 transformed human fibroblasts in the presence of hepatic homogenates and heptachlor epoxide.

Five compounds structurally related to heptachlor epoxide (chlordane, aldrin, dieldrin, heptachlor and chlorendic acid) have produced liver tumors in mice. Chlorendic acid has also produced liver tumors in rats.

CARO -

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : Sufficient evidence exists from rodent studies in which liver carcinomas were induced in two strains of mice of both sexes and in CFN female rats. Several structurally related compounds are liver carcinogens.
- o ORAL SLOPE FACTOR : $9.1E+0$ per (mg/kg)/day
- o DRINKING WATER UNIT RISK : $2.6E-4$ per (ug/L)
- o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
- o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-1 ug/L
E-5 (1 in 100,000)	4E-2 ug/L
E-6 (1 in 1,000,000)	4E-3 ug/L

o ORAL DOSE-RESPONSE DATA :

Tumor Type – hepatocellular carcinomas
 Test Animals – mouse/C3H (Davis); mouse/CD1 (Velsicol)
 Route – diet
 Reference – Davis, 1965; Velsicol, 1973 (see table)

Administered Dose (ppm)	Human Equivalent Dose (mg/kg/day)	Tumor Incidence	Reference
male			
0	0.0	22/73	Davis, 1965
10	0.108	73/79	as diagnosed by Reuber, 1977
female			
0	0.000	2/53	(cited in
10	0.108	77/81	Epstein, 1976)
female			
0	0.00	6/76	Velsicol, 1973
1	0.01	1/70	as evaluated

5	0.052	6/65	by Reuber, 1977
10	0.10	30/57	
male			
0	0.00	0/62	
1	0.01	2/68	
5	0.052	18/68	
10	0.10	52/80	

o ADDITIONAL COMMENTS :

The Davis (1965) study was designed to be for lifetime exposure. Thus, although survival was low, no correction for duration of experiment was made. Five data sets (four in mice and one in rats) show an increased incidence of hepatocellular carcinomas in treated groups compared with controls. There are four slope factors, 27.7 per (mg/kg)/day for C3H male mice, 36.2 per (mg/kg)/day for C3H female mice, 1.04 per (mg/kg)/day for CD-1 female mice, and 6.48 per (mg/kg)/day for CD-1 male mice. Since mice were the more sensitive species tested and to avoid discarding relevant data, the quantitative estimate is based on the geometric mean of 9.1 per (mg/kg)/day. This geometric mean is consistent with the potency estimate from rats of 5.8 per (mg/kg)/day (CFN females).

The above unit risk should not be used if the water concentration exceeds 40 ug/L, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

Adequate numbers of animals were treated in both studies, but survival in the Davis (1985) study was low. A dose-related increase in tumor incidence was observed in CD-1 mice. Slope factors were consistent in two species of rodents.

CARI -

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : Sufficient evidence exists from rodent studies in which liver carcinomas were induced in two strains of mice of both sexes and in CFN female rats. Several structurally related compounds are liver carcinogens.
- o INHALATION UNIT RISK : 2.6E-3 per (ug/cu.m)
- o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
- o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-2 ug/cu.m
E-5 (1 in 100,000)	4E-3 ug/cu.m
E-6 (1 in 1,000,000)	4E-4 ug/cu.m

o INHALATION DOSE-RESPONSE DATA :

The inhalation risk estimates were calculated from the oral data presented in CARO.

o ADDITIONAL COMMENTS :

The above unit risk should not be used if the air concentration exceeds 4 ug/cu.m, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

See CARO.

CARDR-

o CARCINOGENICITY SOURCE :

Source Document – U.S. EPA, 1985, 1986

The values in the 1986 Carcinogenicity Assessment for Chlordane and Heptachlor/Heptachlor Epoxide have been reviewed by the Carcinogen Assessment Group.

DOCUMENT

o REVIEW DATES : 04/01/87
o VERIFICATION DATE : 04/01/87
o EPA CONTACTS :

Dharm V. Singh / OHEA -- (202)260-5958

Jim Cogliano / OHEA -- (202)260-3814

HAONE-

Appropriate data for calculating a One-day HA for heptachlor epoxide are not available. No recommendations are made for the One-day HA.

HATEN-

Appropriate data for calculating a Ten-day HA for heptachlor epoxide are not available. No recommendations are made for the Ten-day HA.

HALTC-

Appropriate data for calculating a Longer-term HA for heptachlor epoxide

are not available. It is recommended that a modified DWEL (adjusted for a 10-kg child) of 0.00013 mg/L (rounded to 0.00015 mg/L) be used as the Longer-term HA.

HALTA-

Appropriate data for calculating a Longer-term HA for heptachlor epoxide are not available. It is recommended that the DWEL of 0.00044 mg/L (rounded to 0.0005 mg/L) be used as the Longer-term HA for the 70-kg adult.

HALIF-

DWEL = 4.4E-4 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date = 09/16/86 (see RDO)

Lifetime HA -- None

Heptachlor epoxide is considered to be a probable human carcinogen. Refer to CAR for information on the carcinogenicity of this substance.

Principal Study -- Dow Chemical Co., 1958 (This study was used in the derivation of the chronic oral RfD; see RDO)

OLEP -

No data available

ALAB -

Determination of heptachlor epoxide is by a liquid-liquid extraction gas chromatographic procedure.

TREAT-

Treatment techniques capable of removing heptachlor epoxide from drinking water include adsorption by granular activated carbon and ozone or ozone/ultra-violet oxidation.

HADR -

o HEALTH ADVISORY SOURCE :

U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Heptachlor Epoxide. Office of Drinking Water, Washington, DC.
DOCUMENT

o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1985.

Public review of HAs following notification of availability in October, 1985.

Scientific Advisory Panel review of HAs in January, 1986.

o EPA DRINKING WATER CONTACT :

Jennifer Orme Zavaleta / OST – (202)260-7586

Edward V. Ohanian / OST – (202)260-7571

CAA - NO DATA

WQCHU-

Water and Fish Consumption: $2.8E-4$ ug/L

Fish Consumption Only: $2.9E-4$ ug/L

Considers technological or economic feasibility? – NO

Discussion – The WQC of $2.8E-4$ ug/L represents a cancer risk level of $1E-6$ based on consumption of contaminated aquatic organisms and water. A WQC of $2.9E-4$ ug/L has also been established based on consumption of contaminated aquatic organisms alone. The heptachlor criteria for both aquatic life and human health serve as the bases for the heptachlor epoxide criteria. The Office of Water has not developed criteria specifically for heptachlor epoxide.

Reference – 45 FR 79318 (11/28/80)

EPA Contact – Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute – $5.2E-1$ ug/L (24-hour average)

Chronic – $3.8E-3$ ug/L

Marine:

Acute – $5.3E-2$ ug/L (24-hour average)

Chronic – $3.6E-3$ ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Water quality criteria for the protection of aquatic life are derived from a minimum data base of acute and chronic tests on a variety of aquatic organisms. The data are assumed to be statistically representative and are used to calculate concentrations which will not have significant short- or long-term effects on 95% of the organisms exposed. Recent criteria (1985 and later) contain duration and frequency stipulations: the acute criteria maximum concentration is a 1-hour average and the chronic criteria continuous concentration is a 4-day average; these averages are not to be exceeded more than once every 3 years, on the average (Stephen et al., 1985). Earlier criteria (1980-1984) contained instantaneous acute and 24-hour average chronic concentrations which were not to be exceeded. These criteria are for heptachlor, rather than heptachlor epoxide.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- EPA has set a MCLG of zero for heptachlor epoxide based on evidence of carcinogenic effects (B2).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.0002 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- EPA has set a MCL equal to the PQL of 0.0002 mg/L, which is associated with a lifetime individual risk of 0.5 E-4.

Monitoring requirements -- All systems monitored for four consecutive quarters every 3 year; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Microextraction/gas chromatography (EPA 505);
electron-capture/gas chromatography (EPA 508); gas chromatographic/mass

spectrometry (EPA 525): PQL= 0.0002 mg/L.

Best available technology -- Granular activated carbon.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___ IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___ IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

SMCL - NO DATA

FISTD-

Status -- Issued (1986)

Reference -- Heptachlor Pesticide Registration Standard. December, 1986
(NTIS No. PB87-175808).

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

Action -- Cancellation of many uses (1988)

Considers technological or economic feasibility? -- YES

Summary of regulatory action -- Based on concern for oncogenicity.

Reference -- 43 FR 12372 (03/24/87; 52 FR 42145 (11/03/87);
53 FR 11798 (04/08/88)

EPA Contact -- Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for heptachlor epoxide is one pound based on potential carcinogenicity. Available data indicate a hazard ranking of high, based on a potency factor of 289.93/mg/kg/day and a weight-of-evidence group B2.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Dow Chemical Company. 1958. MRID No. 00061912. Available from EPA.
Write to FOI, EPA, Washington, DC 20460.
OREF - Dow Chemical Company. 1959a. MRID No. 00062676. Available from EPA.
Write to FOI, EPA, Washington, DC 20460.
OREF - Dow Chemical Company. 1959b. MRID No. 00061911. Available from EPA.
Write to FOI, EPA, Washington, DC 20460.
OREF - Dow Chemical Company. 1966. MRID No. 00086208. Available from EPA.
Write to FOI, EPA, Washington, DC 20460.
OREF - Dow Chemical Company. 1967. MRID No. 00147057. Available from EPA.
Write to FOI, EPA, Washington, DC 20460.
OREF - Dow Chemical Company. 1973a. MRID No. 00050058. Available from EPA.
Write to FOI, EPA, Washington, DC 20460.
OREF - Dow Chemical Company. 1973b. MRID No. 000523262, 00062678, 00064943.

Available from EPA. Write to FOI, EPA, Washington, DC 20460.

IREF - None

CREF - Davis, K.J. 1965. Pathology Report on Mice Fed Aldrin, Dieldrin, Heptachlor and Heptachlor Epoxide for Two Years. Internal FDA memorandum to Dr. A.J. Lehman, July 19.

CREF - Epstein, S.S. 1976. Carcinogenicity of heptachlor and chlordane. Sci. Total Environ. 6: 103-154.

CREF - Reuber, M.D. 1977. Histopathology of carcinomas of the liver in mice ingesting heptachlor or heptachlor epoxide. Exp. Cell Biol. 45: 147-157.

CREF - U.S. EPA. 1985. Hearing Files on Chlordane, Heptachlor Suspension (unpublished draft). Available for inspection at: U.S. EPA, Washington, DC.

CREF - U.S. EPA. 1986. Carcinogenicity Assessment of Chlordane and Heptachlor/Heptachlor Epoxide. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC. OHEA-C-204.

CREF - Velsicol Chemical Corporation. 1973. MRID No. 00062678. Available from EPA. Write to FOI, EPA, Washington, D.C. 20460.

HAREF - Dow Chemical Company. 1958. MRID No. 00061912. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

HAREF - U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Heptachlor Epoxide. Office of Drinking Water, Washington, DC.

Dieldrin

1 - IRIS
NAME - Dieldrin
RN - 60-57-1
IRSN - 221
DATE - 930701
UPDT - 07/01/93, 4 fields
STAT - Oral RfD Assessment (RDO) on-line 09/01/90
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 07/01/93
STAT - Drinking Water Health Advisories (DWHA) on-line 09/01/90
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/07/88 RDO Oral RfD summary on-line
IRH - 09/07/88 CAR Carcinogen summary on-line
IRH - 03/01/90 CAREV Ditraglia citation clarified
IRH - 03/01/90 CAREV Reuber citation year and Deichman spelling corrected
IRH - 03/01/90 CAREV Shirasu citation year corrected
IRH - 03/01/90 CARO Reuber citation year corrected
IRH - 03/01/90 REFS Bibliography on-line
IRH - 04/01/90 CREF Treon and Cleveland, 1955 citation corrected
IRH - 09/01/90 RDO Text edited
IRH - 09/01/90 CAR Text edited
IRH - 09/01/90 HADV Health Advisory on-line
IRH - 09/01/90 REFS Health Advisory references added
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 01/01/92 EXSR Regulatory Action section on-line
IRH - 07/01/93 CARDR Secondary contact's phone number changed
RLEN - 26908
SY - ALVIT
SY - COMPOUND 497
SY - DIELDREX
SY - Dieldrin
SY - DIELDRINE
SY - DIELDRITE
SY - 1,4:5,8-DIMETHANONAPHTHALENE,
1,2,3,4,10,10-HEXACHLORO-6,7-EPOXY-1,4,4a,5,6,7,
SY - 8,8a-OCTAHYDRO, endo,exo-
SY - ENT 16,225
SY - HEOD
SY - HEXACHLOROEOPOXYOCTAHYDRO-endo,exo-DIMETHANONAPHTHALENE
SY - 3,4,5,6,9,9-HEXACHLORO-1a,2,2a,3,6,6a,7,7a-OCTAHYDRO-2,7:3,6-DIMETHANON
APHTH
SY - (2,3-b)OXIRENE
SY - ILLOXOL
SY - NA 2761
SY - NCI-C00124
SY - OCTALOX
SY - PANORAM D-31
SY - QUINTOX
SY - RCRA WASTE NUMBER P037

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver lesions	NOAEL: 0.1 ppm (0.005 mg/kg/day)	100	1	5E-5 mg/kg/day
2-Year Rat Feeding Study	LOAEL: 1.0 ppm (0.05 mg/kg/day)			
Walker et al., 1969				

*Conversion Factors: 1 ppm = 0.05 mg/kg/day (assumed rat food consumption)

o ORAL RFD STUDIES :

Walker, A.I.T., D.E. Stevenson, J. Robinson, R. Thorpe and M. Roberts. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two-year oral exposures of rats and dogs. Toxicol. Appl. Pharmacol. 15: 345-373.

Walker et al. (1969) administered dieldrin (recrystallized, 99% active ingredient) to Carworth Farm "E" rats (25/sex/dose; controls 45/sex) for 2 years at dietary concentrations of 0, 0.1, 1.0, or 10.0 ppm. Based on intake assumptions presented by the authors, these dietary levels are approximately equal to 0, 0.005, 0.05 and 0.5 mg/kg/day. Body weight, food intake, and general health remained unaffected throughout the 2-year period, although at 10.0 ppm (0.5 mg/kg/day) all animals became irritable and exhibited tremors and occasional convulsions. No effects were seen in various hematological and clinical chemistry parameters. At the end of 2 years, females fed 1.0 and 10.0 ppm (0.05 and 0.5 mg/kg/day) had increased liver weights and liver-to-body weight ratios ($p < 0.05$). Histopathological examinations revealed liver parenchymal cell changes including focal proliferation and focal hyperplasia. These hepatic lesions were considered to be characteristic of exposure to an organochlorine insecticide. The LOAEL was identified as 1.0 ppm (0.005 mg/kg/day) and the NOAEL as 0.1 ppm (0.005 mg/kg/day).

o ORAL RFD UNCERTAINTY :

UF – The UF of 100 allows for uncertainty in the extrapolation of dose levels from laboratory animals to humans (10A) and uncertainty in the threshold for sensitive humans (10H).

o ORAL RFD MODIFYING FACTOR :

MF = None

o ORAL RFD COMMENTS :

Data considered for establishing the RfD:

1) 2-Year Feeding - rat: Principal study - see previous description

2) 2-Year Feeding (oncogenic) - dog: Systemic NOEL=0.005 mg/kg/day; LEL= 0.05 mg/kg/day (increased liver weight and liver/body weight ratios, increased plasma alkaline phosphatase, and decreased serum protein concentration) (Walker et al., 1969)

3) 2-Year Feeding - rat: Systemic LEL=0.5 ppm (approximately 0.025 mg/kg/day),

(liver enlargement with histopathology); (Fitzhugh et al., 1964)

4) 2-Year Feeding (oncogenic) - mouse: Systemic LEL=0.1 ppm (0.015 mg/kg/day), (liver enlargement with histopathology); (Walker et al., 1972)

5) 25-Month Feeding - dog: Systemic NOEL=0.2 mg/kg/day; LEL=0.5 mg/kg/day, (weight loss and convulsions); (Fitzhugh et al., 1964)

6) Teratology - mouse: Teratogenic NOEL=6.0 mg/kg/day (HDT, gestational days 7-16); Maternal LEL=6.0 mg/kg/day (HDT, decrease in maternal weight gain); Fetotoxic LEL=6.0 mg/kg/day (HDT, decreased numbers of caudal ossification centers and increases in supernumerary ribs); (Chernoff et al., 1975). This study was not considered since 41% of the test dams died at the highest dose tested.

o ORAL RFD CONFIDENCE :

Study -- Low
Data Base -- Medium
RfD -- Medium

The principal study is an older study for which detailed data are not available and in which a wide range of doses was tested. The chronic toxicity evaluation is relatively complete and supports the critical effect, if not the magnitude of effects. Reproductive studies are lacking. The RfD is given a medium confidence rating because of the support for the critical effect from other dieldrin studies, and from studies on organochlorine insecticides in general.

o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1987

Other EPA Documentation -- None

o REVIEW DATES : 04/16/87
o VERIFICATION DATE : 04/16/87
o EPA CONTACTS :

Krishan Khanna / OST -- (202)260-7588

Henry Spencer / OST -- (202)557-4383

RDI - NO DATA
CAREV-

o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Dieldrin is carcinogenic in seven strains of mice when administered orally. Dieldrin is

structurally related to compounds (aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid) which produce tumors in rodents.

o HUMAN CARCINOGENICITY DATA :

Inadequate. Two studies of workers exposed to aldrin and to dieldrin reported no increased incidence of cancer. Both studies were limited in their ability to detect an excess of cancer deaths. Van Raalte (1977) observed two cases of cancer (gastric and lymphosarcoma) among 166 pesticide manufacturing workers exposed 4-19 years and followed from 15-20 years. Exposure was not quantified, and workers were also exposed to other organochlorine pesticides (endrin and telodrin). The number of workers studied was small, the mean age of the cohort (47.7 years) was young, the number of expected deaths was not calculated, and the duration of exposure and of latency was relatively short.

In a retrospective mortality study, Ditraglia et al. (1981) reported no statistically significant excess in deaths from cancer among 1155 organochlorine pesticide manufacturing workers [31 observed vs. 37.8 expected, Standardized Mortality Ratio (SMR) = 82]. Workers were employed for 6 months or more and followed 13 years or more (24,939 person-years). Workers with no exposure (for example, office workers) were included in the cohort. Vital status was not known for 112 or 10% of the workers, and these workers were assumed to be alive; therefore additional deaths may have occurred but were not observed. Exposure was not quantified and workers were also exposed to other chemicals and pesticides (including endrin). Increased incidences of deaths from cancer were seen at several specific sites: esophagus (2 deaths observed, SMR = 235); rectum (3, SMR = 242); liver (2, SMR = 225); and lymphatic and hematopoietic system (6, SMR = 147), but these site-specific incidences were not statistically significantly increased.

o ANIMAL CARCINOGENICITY DATA :

Sufficient. Dieldrin has been shown to be carcinogenic in various strains of mice of both sexes. At different dose levels the effects range from benign liver tumors, to hepatocarcinomas with transplantation confirmation, to pulmonary metastases.

The Food and Drug Administration (FDA) conducted a long-term carcinogenesis bioassay for dieldrin (Davis and Fitzhugh, 1962). Ten ppm dieldrin was administered orally to 218 male and female C3HeB/Fe mice for 2 years. The study was compromised by the poor survival rate, lack of detailed pathology, loss of a large percentage of the animals to the study, and failure to treat the data for males and females separately. A statistically significant increase in incidence of hepatomas was observed in the treated groups versus the control groups in both males and females. In FDA follow-up study, Davis (1965) examined 100 male and 100 female C3H mice which had been orally administered 10 ppm dieldrin. The same limitations as the previous study were reported. The incidence of benign hepatomas and hepatic carcinomas was significantly increased in the dieldrin group. A reevaluation of the histological material of both studies was done by Reuber in 1974 (Epstein, 1975a,b; 1976). He concluded that the hepatomas were malignant and that dieldrin was hepatocarcinogenic for male and female C3HeB/Fe and C3H mice.

Walker et al. (1972) conducted several studies of dieldrin in CF1 mice of both sexes. Dieldrin was administered orally at concentrations of 0, 0.1, 1.0, and 10 ppm. Treatment groups varied from 87 to 288 animals of each sex. Surviving animals were sacrificed during weeks 132-140. Incidence of tumors was related to the number of dose levels and the dose administered. Effects were detected at the lowest dieldrin level tested (0.1 ppm) in both male and female mice. Dieldrin also produced significant increases (<0.05) in the incidence of pulmonary adenomas, pulmonary carcinomas, lymphoid tumors, and "other" tumors in female mice.

Diets containing 10 ppm dieldrin were fed to groups of 30 CF1 mice of both sexes for 110 weeks (Thorpe and Walker, 1973). The control group consisted of 45 mice of both sexes. A statistically significant increase ($p<0.01$) in incidence of liver tumors was found in both sexes of treated animals relative to controls. The liver tumors appeared much earlier in treated animals than controls.

Technical-grade dieldrin ($>96\%$) was fed to B6C3F1 mice (50/sex/dose) at TWA doses of 0, 2.5, or 5 ppm for 80 weeks followed by an observation period of 10 to 13 weeks (NCI, 1978a). Matched control groups consisted of 20 untreated males and 10 untreated females. No significant difference in survival was noted. A significant dose-related increase in hepatocellular carcinoma was found in male mice when compared with pooled controls.

Tennekes et al. (1981) fed groups of 19 to 82 male CF1 mice control or dieldrin-supplemented (10 ppm) diets or control diets for 110 weeks. Dieldrin produced a statistically significant increased incidence of hepatocellular carcinomas in the treated group.

Dieldrin ($>99\%$) was continuously fed in the diet for 85 weeks to 50 C3H/He, 62 B6C3F1, and 71 C57Bl/6J male mice (Meierhenry et al., 1983). Controls were 50 to 76 males of each strain. Dieldrin produced a significant increase in the incidence of hepatocellular carcinomas compared with controls in all three strains.

Seven studies with four strains of rats fed 0.1 to 285 ppm dieldrin varying in duration of exposure from 80 weeks to 31 months did not produce positive results for carcinogenicity (Treon and Cleveland, 1955; Fitzhugh et al., 1964; Song and Harville, 1964; Walker et al., 1969; Deichmann et al., 1970; NCI, 1978a,b). Three of these studies used Osborne-Mendel rats, two studies used Carworth rats, and one each used Fischer 344 and Holtzman strains. Only three of the seven studies are considered adequate in design and conduct. The others used too few animals, had unacceptably high levels of mortality, were too short in duration, and/or had inadequate pathology examination or reporting.

o SUPPORTING DATA :

Dieldrin causes chromosomal aberrations in mouse cells (Markaryan, 1966; Majumdar et al., 1976) and in human lymphoblastoid cells (Trepanier et al., 1977), forward mutation in Chinese hamster V79 cells (Ahmed et al., 1977), and unscheduled DNA synthesis in rat (Probst et al., 1981) and human cells (Rocchi et al., 1980). Dieldrin did not produce responses in 13 other mutagenicity tests. Negative responses were given in assays for gene conversion in *S.*

cerevisiae, back-mutation in *S. marcesans*, forward mutation (Gal Rz2 in *E. coli*), and forward mutation to streptomycin resistance in *E. coli* (Fahrig, 1974). Negative responses were produced in reverse mutation assays with six strains of *S. typhimurium* with or without metabolic activation (Bidwell et al., 1975; Marshall et al., 1976; Shirasu et al., 1976; Wade et al., 1979; Haworth et al., 1983). Majumdar et al. (1977), however, reported that dieldrin was mutagenic for *S. typhimurium* with and without metabolic activation.

Five compounds structurally related to dieldrin - aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorondic acid - have induced malignant liver tumors in mice. Chlorendic acid has also induced liver tumors in rats.

CARO -

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : Dieldrin is carcinogenic in seven strains of mice when administered orally. Dieldrin is structurally related to compounds (aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid) which produce tumors in rodents.
- o ORAL SLOPE FACTOR : $1.6E+1$ per (mg/kg)/day
- o DRINKING WATER UNIT RISK : $4.6E-4$ per (ug/L)
- o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
- o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-1 ug/L
E-5 (1 in 100,000)	2E-2 ug/L
E-6 (1 in 1,000,000)	2E-3 ug/L

o ORAL DOSE-RESPONSE DATA :

Tumor Type -- liver carcinoma
 Test Animals -- mouse
 Route -- diet
 Reference -- see table

Sex/Strain Slope Factor Reference

Male, C3H	22	Davis (1965), reevaluated by Reuber, 1974 (cited in Epstein, 1975a)
Female, C3H	25	Davis (1965), reevaluated by Reuber, 1974 (cited

in Epstein, 1975a)

Male, CF1	25	Walker et al. (1972)
Female, CF1	28	Walker et al. (1972)
Male, CF1	15	Walker et al. (1972)
Female, CF1	7.1	Walker et al. (1972)
Male, CF1	55	Thorpe and Walker (1973)
Female, CF1	26	Thorpe and Walker (1973)
Male, B6C3F1	9.8	NCI (1978a,b)
Male, CF1	18	Tennekes et al. (1981)
Male, C57B1/6J	7.4	Meierhenry et al. (1983)
Male, C3H/He	8.5	Meierhenry et al. (1983)
Male, B6C3F1	11	Meierhenry et al. (1983)

o ADDITIONAL COMMENTS :

The slope factor is the geometric mean of 13 slope factors calculated from liver carcinoma data in both sexes of several strains of mice. Inspection of the data indicated no strain or sex specificity of carcinogenic response.

The unit risk should not be used if the water concentration exceeds 20 ug/L, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

The individual slope factors calculated from 13 independent data sets range within a factor of 8.

CARI -

o CLASSIFICATION : B2; probable human carcinogen

o BASIS FOR CLASSIFICATION : Dieldrin is carcinogenic in seven strains of mice when administered orally. Dieldrin is structurally related to compounds (aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid) which produce tumors in rodents.

o INHALATION UNIT RISK : 4.6E-3 per (ug/cu.m)

o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk

o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-2 ug/cu.m
E-5 (1 in 100,000)	2E-3 ug/cu.m
E-6 (1 in 1,000,000)	2E-4 ug/cu.m

o INHALATION DOSE-RESPONSE DATA :

Calculated from oral data in CARO.

o ADDITIONAL COMMENTS :

The unit risk should not be used if air concentrations exceed 2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

This inhalation risk estimate was based on oral data.

CARDR-

o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1986
DOCUMENT

- o REVIEW DATES : 03/05/87
- o VERIFICATION DATE : 03/05/87
- o EPA CONTACTS :

Dharm Singh /OHEA -- (202)260-5958

Jim Cogliano / OHEA -- (202)260-3814

HAONE-

Appropriate data for calculating a One-day HA are not available. It is recommended that the modified DWEL of 0.0005 mg/L be used as the One-day HA.

HATEN-

Appropriate data for calculating a Ten-day HA are not available. It is recommended that the modified DWEL of 0.0005 mg/L be used as the Ten-day HA.

HALTC-

Appropriate data for calculating Longer-term HAs for dieldrin are not available. It is recommended that the modified DWEL of 0.0005 mg/L be used as the Longer-term HA for the 10-kg child.

HALTA-

Appropriate data for calculating Longer-term HAs for dieldrin are not available. It is recommended that the modified DWEL of 0.002 mg/L be used as the Longer-term HA for the 70-kg adult.

HALIF-

Drinking Water Equivalent Level (DWEL) -- 2E-3 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date -- 04/16/87 (see Section I.A. in this file)

Lifetime HA -- None

Dieldrin is considered to be a probable human carcinogen. Lifetime HAs are not recommended for known or probable human carcinogens. The estimated excess cancer risk associated with lifetime exposure to drinking water containing dieldrin at the DWEL of 2 ug/L is approximately 8.05×10^{-4} . Refer to Section II for the carcinogenicity assessment for dieldrin.

Principal Study -- Walker et al., 1969 (This study was used in the derivation of the chronic oral RfD; see RDO)

OLEP -

The odor threshold for dieldrin in water is reported as 0.04 mg/L.

ALAB -

Determination of dieldrin is by a liquid-liquid extraction gas chromatographic procedure.

TREAT-

Available data indicate that reverse osmosis, granular activated carbon

adsorption, ozonation, and conventional treatment will remove dieldrin from water.

HADR -

o HEALTH ADVISORY SOURCE :

U.S. EPA. 1989. Drinking Water Health Advisories: Pesticides. Lewis Publishers, Chelsea, MI. p. 299-312.

DOCUMENT

o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1987.

Public review of HAs in January-March 1988.

o EPA DRINKING WATER CONTACT :

Krishan Khanna / OST -- (202)260-7588

Edward V. Ohanian / OST -- (202)260-7571

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 7.1E-5 ug/L

Fish Consumption Only: 7.6E-5 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient concentration should be zero. However, zero may not be attainable at this time, so the recommended criterion represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 1.0E+0 ug/L
Chronic -- 1.9E-3 ug/L

Marine:

Acute -- 7.1E-1 ug/L
Chronic -- 1.9E-3 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

No data available

MCL -

No data available

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (Proposed, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Microextraction/gas chromatography (EPA 505);
electron-capture/gas chromatography (EPA 508); gas chromatographic/mass

spectrometry (EPA 525).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

SMCL - NO DATA

FISTD-

No data available

FIREV-

Action -- Registration canceled (1974)

Considers technological or economic feasibility? -- NO

Summary of regulatory action -- Cancellation of all but termiticide and use. Criteria of concern: carcinogenicity, bio-accumulation, hazard to wildlife, and other chronic effects.

Reference -- 39 FR 37246 (10/18/74)

EPA Contact -- Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for dieldrin is based on aquatic toxicity as established under CWA Section 311 (40 CFR 117.3) and potential carcinogenicity. The available data indicate that the aquatic 96-Hour Median threshold Limit is less than 0.1 ppm, which corresponds to an RQ of 1 pound. Available data also indicate a hazard ranking of high and a weight of evidence classification of Group B2, which corresponds to an RQ of 1 pound.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Chernoff, N., R.J. Kavlock, J.R. Kathrein, J.M. Dunn and J.K. Haseman. 1975. Prenatal effects of dieldrin and photodieldrin in mice and rats. *Toxicol. Appl. Pharmacol.* 31: 302-308.

OREF - Fitzhugh, O.G., A.A. Nelson and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. *Food Cosmet. Toxicol.* 2: 551-562.

OREF - U.S. EPA. 1987. *Dieldrin: Health Advisory. Office of Drinking Water*, Washington, DC. NTIS PB 88-113543/AS.

OREF - Walker, A.I.T., D.E. Stevenson, J. Robinson, E. Thorpe and M. Roberts. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two-year oral exposures of rats and dogs. *Toxicol. Appl. Pharmacol.* 15: 345-373.

OREF - Walker, A.I.T., E. Thorpe and D.E. Stevenson. 1972. The toxicology of dieldrin (HEOD). I. Long-term oral toxicity studies in mice. *Food Cosmet. Toxicol.* 11: 415-432.

IREF - None

CREF - Ahmed, F.E., R.W. Hart and N.J. Lewis. 1977. Pesticide induced DNA damage and its repair in cultured human cells. *Mutat. Res.* 42: 161-174.

CREF - Bidwell, K., E. Weber, I. Neinholt, T. Connor and M.S. Legator. 1975. Comprehensive evaluation for mutagenic activity of dieldrin. *Mutat. Res.* 31: 314. (Abstract)

CREF - Davis, K.J. 1965. Pathology report on mice fed aldrin, dieldrin, heptachlor or heptachlor epoxide for two years. Internal FDA memorandum to Dr. A.J. Lehman. July 19. (Cited in: U.S. EPA, 1986)

CREF - Davis, K.J. and O.G. Fitzhugh. 1962. Tumorigenic potential of aldrin and dieldrin for mice. *Toxicol. Appl. Pharmacol.* 4: 187-189.

CREF - Deichmann, W.B., W.E. MacDonald, E. Blum, et al. 1970. Tumorigenicity of aldrin, dieldrin and endrin in the albino rat. *Ind. Med. Surg.* 39: 426-434.

- CREF - Ditraglia, D., D.P. Brown, T. Namekata and M. Iverson. 1981. Mortality study of workers employed at organochlorine pesticide manufacturing plants. *Scand. J. Work. Env. Health*. 7 (Suppl. 4): 140-146.
- CREF - Epstein, S.S. 1975a. The carcinogenicity of dieldrin. Part 1. *Sci. Total Environ.* 4: 1-52.
- CREF - Epstein, S.S. 1975b. The carcinogenicity of dieldrin. Part 2. *Sci. Total Environ.* 4: 205-217.
- CREF - Epstein, S.S. 1976. Case study 5: Aldrin and dieldrin suspension based on experimental evidence and evaluation and societal needs. *Ann. NY. Acad. Sci.* 271: 187-195.
- CREF - Fahrig, R. 1974. Comparative mutagenicity studies with pesticides. IARC Scientific Press No. 10.
- CREF - Fitzhugh, O.G., A.A. Nelson and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. *Food Cosmet. Toxicol.* 2: 551-562.
- CREF - Haworth, S., T. Lawlor, K. Mortelmans, W. Speck and E. Zeigler. 1983. Salmonella mutagenicity test results for 250 chemicals. *Environ. Mutag.* 5(Suppl. 1): 1-142.
- CREF - Majumdar, S.K., H.A. Kopelman and M.J. Schnitman. 1976. Dieldrin-induced chromosome damage in mouse bone-marrow and WI-38 human lung cells. *J. Hered.* 67: 303-307.
- CREF - Majumdar, S.K., L.G. Maharam and G.A. Viglianti. 1977. Mutagenicity of dieldrin in the Salmonella-microsome test. *J. Hered.* 68: 184-185.
- CREF - Markaryan, D.S. 1966. Cytogenic effect of some chlorinated insecticides on mouse bone-marrow cell nuclei. *Soviet Genetics*. 2(1): 80-82.
- CREF - Marshall, T.C., H.W. Dorough and H.E. Swim. 1976. Screening of pesticides for mutagenic potential using Salmonella typhimurium mutants. *J. Agric. Chem.* 24: 560-563.
- CREF - Meierhenry, E.F., B.H. Reuber, M.E. Gershwin, L.S. Hsieh and S.W. French. 1983. Dieldrin-induced Mallory bodies in hepatic tumors of mice of different strains. *Hepatology*. 3: 90-95.
- CREF - NCI (National Cancer Institute). 1978a. Bioassays of aldrin and dieldrin for possible carcinogenicity. DHEW Publication No. (NIH) 78-821. National Cancer Institute Carcinogenesis Technical Report Series, No. 21. NCI-CG-TR-21.
- CREF - NCI (National Cancer Institute). 1978b. Bioassays of aldrin and dieldrin for possible carcinogenicity. DHEW Publication No. (NIH) 78-822. National Cancer Institute Carcinogenesis Technical Report Series, No. 22. NCI-CG-TR-22.
- CREF - Probst, G.S., R.E. McMahon, L.W. Hill, D.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 chemicals. *Environ. Mutagen.* 3: 11-32.
- CREF - Reuber, M.D. 1974. Exhibit 42. Testimony at hearings on aldrin/dieldrin. (Cited in: Epstein, 1975a)
- CREF - Rocchi, P., P. Perocco, W. Alberghini, A. Fini and G. Prodi. 1980. Effect of pesticides on scheduled and unscheduled DNA synthesis of rat thymocytes and human lymphocytes. *Arch. Toxicol.* 45: 101-108.
- CREF - Shirasu, Y., M. Moriya, K. Kato, A. Furuhashi and T. Kada. 1976. Mutagenicity screening of pesticides in the microbial system. *Mutat. Res.* 40(1): 19-30.
- CREF - Song, J. and W.E. Harville. 1964. Carcinogenicity of aldrin and dieldrin in mouse and rat liver. *Fed. Proc. Fed. Am. Soc. Exp. Biol.* 23: 336.

CREF - Tennekes, H.A., A.S. Wright, K.M. Dix and J.H. Koeman. 1981. Effects of dieldrin, diet, and bedding on enzyme function and tumor incidence in livers of male CF-1 mice. Cancer Res. 41: 3615-3620.

CREF - Thorpe, E. and A.I.T. Walker. 1973. The toxicology of dieldrin (HEOD).
Part

HAREF- NO DATA

Beryllium

1 - IRIS
 NAME - Beryllium
 RN - 7440-41-7
 IRSN - 11
 DATE - 930201
 UPDT - 02/01/93, 1 field
 STAT - Oral RfD Assessment (RDO) on-line 02/01/93
 STAT - Inhalation RfC Assessment (RDI) no data
 STAT - Carcinogenicity Assessment (CAR) on-line 09/01/92
 STAT - Drinking Water Health Advisories (DWHA) no data
 STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
 IRH - 03/01/88 RDO Reference dose table clarified
 IRH - 03/01/88 RDO Text added
 IRH - 09/07/88 CAR Carcinogen summary on-line
 IRH - 01/01/90 CAREV References clarified
 IRH - 01/01/90 CAREV Text revised
 IRH - 01/01/90 CARO Quantitative estimate for oral exposure section added
 IRH - 01/01/90 CARI Text revised
 IRH - 01/01/90 CARDR Work group review dates and verification date added
 IRH - 01/01/90 REFS Bibliography on-line
 IRH - 02/01/90 OREF Puzanova et al. 1978 citation corrected
 IRH - 02/01/90 CREF Wagner et al. 1969 citation corrected
 IRH - 09/01/90 RDO Morgareidge ref. now Cox (same study-authors reversed)
 IRH - 09/01/90 RCRA EPA contact changed
 IRH - 09/01/90 OREF Morgareidge ref. now Cox (same study-authors reversed)
 IRH - 01/01/91 CAR Text edited
 IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
 IRH - 01/01/92 EXSR Regulatory actions updated
 IRH - 09/01/92 CAREV U.S. EPA citation year corrected, paragraph 3
 IRH - 09/01/92 CARDR Source document year corrected
 IRH - 09/01/92 CARDR Review statement revised
 IRH - 09/01/92 CREF U.S. EPA reference year corrected
 IRH - 02/01/93 RDO Primary contact changed
 RLEN - 27537
 SY - Beryllium
 SY - Beryllium-9
 SY - Glucinum
 SY - RCRA waste number P015
 SY - UN 1567

RDO -

o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
No adverse effects	NOAEL: 5 ppm in drinking water (0.54 mg/kg bw/day)	100	1	5E-3
Rat, Chronic Oral Bioassay				
Schroeder and Mitchner, 1975	LOAEL: none			

*Conversion Factors: 5 ppm (5 mg/L) x 0.035 L/day / 0.325 kg bw = 0.54 mg/kg

bw/day

o ORAL RFD STUDIES :

Schroeder, H.A. and M. Mitchner. 1975. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. J. Nutr. 105: 421-427.

Fifty-two weanling Long-Evans rats of each sex received 0 or 5 ppm beryllium (as BeSO₄, beryllium sulfate) in drinking water. Exposure was for the lifetime of the animals. At natural death the rats were dissected and gross and microscopic changes were noted in heart, kidney, liver, and spleen. There were no effects of treatment on these organs or on lifespan, urinalysis, serum glucose, cholesterol, and uric acid, or on numbers of tumors. Male rats experienced decreased growth rates from 2 to 6 months of age.

Similar studies were carried out on Swiss (CD strain) mice in groups of 54/sex at doses of approximately 0.95 mg/kg/day (Schroeder and Mitchner, 1975). Female animals showed decreased body weight compared with untreated mice at 6 of 8 intervals. Male mice exhibited slight increases in body weight. These effects were not considered adverse, therefore, 0.95 mg/kg/day is considered a NOAEL.

An unpublished investigation by Cox et al. (1975) indicates a much higher dose level (approximately 25 mg/kg/day) in the diet may be a NOEL.

o ORAL RFD UNCERTAINTY :

UF -- The uncertainty factor of 100 reflects a factor of 10 each for interspecies conversion and for the protection of sensitive human subpopulations.

o ORAL RFD MODIFYING FACTOR :

MF -- None

o ORAL RFD COMMENTS :

This RfD is limited to soluble beryllium salts. Data on the teratogenicity or reproductive effects of beryllium are limited. It has been reported to produce embryoletality and terata in chick embryos (Puzanova et al., 1978).

o ORAL RFD CONFIDENCE :

Study -- Low
Data Base -- Low
RfD -- Low

Confidence in the study is rated as low because only one dose level was administered. Although numerous inhalation investigations and a supporting chronic oral bioassay in mice exist, along with the work by Cox et al. (1975) which indicates that a higher dose level might be a NOEL, these studies are considered as low to medium quality; thus, the data base is given a low confidence rating. The overall confidence in the RfD is low, reflecting the need for more toxicity data by the oral route.

o ORAL RFD SOURCE DOCUMENT :

Source Document – U.S. EPA, 1985

The 1985 Drinking Water Criteria Document for Beryllium is currently undergoing Agency review.

o REVIEW DATES : 12/02/85
o VERIFICATION DATE : 12/02/85
o EPA CONTACTS :

Linda R. Papa / OHEA – (513)569-7587

Krishan Khanna / OST – (202)260-7588

RDI - NO DATA

CAREV-

o CLASSIFICATION : B2; probable human carcinogen.
o BASIS FOR CLASSIFICATION : Beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.

o HUMAN CARCINOGENICITY DATA :

Inadequate. Reported increases, while apparently associated with exposure, did not take a variety of possible confounding factors into account.

Wagoner et al. (1980) observed 47 deaths from cancer among 3055 white males employed in beryllium-processing with a median duration of employment of 7.2 months. Among the 2068 followed for 25 years or more, 20 lung cancer deaths were observed. These increased incidences were statistically significant. When lung cancer mortality data became available for 1968-1975, the number of expected deaths was recalculated and the increased incidence was statistically significant only among workers followed 25 years or more (Bayliss, 1980; MacMahon, 1977, 1978). When the number of expected deaths was adjusted for smoking, the increased incidence was no longer significant (U.S. EPA, 1986).

An earlier study of workers from this same beryllium processing plant, and several studies of workers from this plant combined with workers from other beryllium plants, have reported a statistically significant increased incidence of lung cancer (Bayliss and Wagoner, 1977; Mancuso, 1970, 1979, 1980). No adjustment was made for smoking in these studies, and all were limited in their ability to detect a possible increased incidence of lung cancer because of methodological constraints and deficiencies.

o ANIMAL CARCINOGENICITY DATA :

Sufficient. Based on the evidence for induction of tumors by a variety of beryllium compounds in male and female monkeys and in several strains of rats of both sexes, via inhalation and intratracheal instillation, and the induction of osteosarcomas in rabbits by intravenous or intramedullary injection in multiple studies.

Slight increases in cancer incidence (not statistically significant in comparison with controls) were reported in Long-Evans rats (52/sex/group) administered 5 ppm beryllium sulfate in the drinking water for a lifetime. The authors reported a slight excess of grossly observed tumors in the 5 ppm group (9/33) over controls (4/26) in the male rats. The power of this test to detect a carcinogenic effect was reduced by high mortality (approximately 60% survived a pneumonia epidemic at 20 months) (Schroeder and Mitchener, 1975a). Schroeder and Mitchener (1975b) administered 5 ppm beryllium sulfate in drinking water to Swiss mice (54/sex/group) over a lifetime. A non-statistically significant increase in incidence of lymphoma leukemias were reported in the females (9/52) relative to controls (3/47).

An increase in reticulum cell sarcomas of the lungs was seen in male, but not female Wistar-derived rats administered beryllium sulfate in the diet at 5 and 50 ppm, but not at 500 ppm (Morgareidge et al., 1977). The incidence in males equaled 10/49, 17/35, 16/40 and 12/39 for the control, low, intermediate and high dose groups, respectively. Since the results were published only as an abstract, and since no response was seen at the highest dose, these results are considered to be only suggestive for the induction of cancer via this route.

Osteogenic sarcomas were induced in rabbits by intravenous injection of beryllium compounds in at least 12 different studies and by intramedullary injection in at least four studies (U.S. EPA, 1991). Bone tumors were induced by beryllium oxide, zinc beryllium silicate, beryllium phosphate, beryllium silicate and beryllium metal. No bone tumors were reported to be induced by intravenous injection of beryllium oxide or zinc beryllium silicate in rats or guinea pigs (Gardner and Heslington, 1946). Positive results, however, were reported in mice injected with zinc beryllium silicate, although the numbers were not listed (Cloudman et al., 1949). The sarcomas were generally reported to be quite malignant and metastasized to other organs.

Lung tumors, primarily adenomas and adenocarcinomas, have been induced via the inhalation route in both male and female Sprague-Dawley rats during exposure periods of up to 72 weeks by beryllium sulfate (Reeves et al., 1967), in both male and female Sherman and Wistar rats by beryllium phosphate, beryllium fluoride and zinc beryllium silicate (Schepers, 1961), in male Charles River CR-CD rats by beryl ore (Wagner et al., 1969) and in both male and female rhesus monkeys by beryllium sulfate (Vorwald, 1968). Positive results were seen in rats exposed to beryllium sulfate at concentrations as low as 2 ug/cu.m (Vorwald, 1968).

Tumors were also induced by intratracheal instillation of metallic beryllium, beryllium-aluminum alloys and beryllium oxide in both Wistar rats and rhesus monkeys. Adenomas, adenocarcinomas and malignant lymphomas were seen in the lungs, with lymphosarcomas and fibrosarcomas present at extrapulmonary sites (Groth et al., 1980; Ishinishi et al., 1980).

o SUPPORTING DATA :

Beryllium sulfate and beryllium chloride have been shown to be nonmutagenic in bacterial and yeast gene mutation assays (Simmon et al., 1979). In contrast, gene mutation studies in Chinese hamster V79 and CHO cells were positive (Miyaki et al., 1979; Hsie et al., 1979). Chromosomal aberrations and sister chromatid exchange were also induced by beryllium in cultured human lymphocytes and Syrian hamster embryo cells (Larramendy et al., 1981).

CARO -

o CLASSIFICATION : B2; probable human carcinogen.

o BASIS FOR CLASSIFICATION : Beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.

o ORAL SLOPE FACTOR : 4.3 per(mg/kg)/day

o DRINKING WATER UNIT RISK : 1.2E-4 per(ug/L)

o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk

o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	8.3E-1 ug/L
E-5 (1 in 100,000)	8.3E-2 ug/L
E-6 (1 in 1,000,000)	8.3E-3 ug/L

o ORAL DOSE-RESPONSE DATA :

Tumor Type -- gross tumors, all sites combined
Test Animals -- rat/Long-Evans, male
Route -- drinking water
Reference -- Schroeder and Mitchener, 1975a

Administered Dose		Human Equivalent Dose		Tumor
ppm	(mg/kg)/day		(mg/kg/day)	Incidence
0	0	0	4/26	
5	0.54	0.09	9/33	

o ADDITIONAL COMMENTS :

The solubility and speciation of beryllium in air and water media vary, with ambient air characterized by relatively insoluble beryllium compounds such as beryllium oxide and metallic beryllium, and water characterized by

more soluble forms. Carcinogenic potency varies according to the form of beryllium present.

Human equivalent doses were calculated using a human body weight of 70 kg, an animal weight of 0.325 kg and length of exposure, experiment and lifespan of 1126 days for treated and control animals.

The unit risk should not be used if the water concentration exceeds $8.3E+1$ ug/L, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

The estimate is derived from a study which did not show a significant increase in tumorigenic response. While this study is limited by use of only one non-zero dose group, the occurrence of high mortality and unspecified type and site of the tumors, it was used as the basis of the quantitative estimate because exposure occurred via the most relevant route. Oral risk estimates derived by extrapolation from studies in other species/strains for the intravenous and inhalation routes (also highly uncertain) are within an order of magnitude.

CARI -

o CLASSIFICATION : B2; probable human carcinogen.

o BASIS FOR CLASSIFICATION : Beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.

o INHALATION UNIT RISK : $2.4E-3$ per (ug/cu.m)

o DOSE EXTRAPOLATION METHOD : Relative risk

o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	$4E-2$ ug/cu.m
E-5 (1 in 100,000)	$4E-3$ ug/cu.m
E-6 (1 in 1,000,000)	$4E-4$ ug/cu.m

o INHALATION DOSE-RESPONSE DATA :

Tumor Type --

Test Animals -- humans

Route -- inhalation, occupational exposure

Reference --

Beryllium			95 percent	
Concentration	Fraction	Effective	Upper-bound	Unit

in Workplace (ug/cu.m)	of Lifetime	dose (ug/cu.m)	Estimate of Relative Risk	Risk /ug/cu.m
100	1.00	21.92	1.98	1.61E-3
			2.09	1.79E-3
	0.25	5.48	1.98	6.44E-3
			2.09	7.16E-3
1000	1.00	219.18	1.98	1.61E-4
			2.09	1.79E-4
	0.25	54.79	1.98	6.44E-4
			2.09	7.16E-4

o ADDITIONAL COMMENTS :

Human data were used for the inhalation exposure quantitation despite limitations in the study. Humans are most likely to be exposed by inhalation to beryllium oxide, rather than other beryllium salts. Animal studies by inhalation of beryllium oxide have utilized intratracheal instillation, rather than general inhalation exposure.

Effective dose was determined by adjusting for duration of daily (8/24 hours) and annual (240/365 days) exposure, and the fraction of the lifetime at risk (i.e., time from onset of employment to termination of follow-up). The risk estimates were based on the data of Wagoner et al. (1980) in which the smoking adjusted, expected lung cancer deaths were found to range from 13.91 to 14.67, in comparison to 20 observed. Relative risk estimates of 1.36 and 1.44 were derived and the 95% confidence limits of these estimates, 1.98 and 2.09, respectively, were used to estimate the lifetime cancer risk. Note that all of the above estimates are based on one data set using a range of estimated exposure and exposure times. Because of uncertainties regarding workplace beryllium concentration and exposure duration, unit risks were derived using two estimates each of concentration, fraction of lifetime exposed and relative risk. The recommended value is the arithmetic mean of the 8 derived unit risks.

The unit risk should not be used if the air concentration exceeds 4 ug/cu.m, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

The estimate of risk for inhalation exposure was based upon an epidemiologic study having several confounding variables. The estimates of exposure levels and duration were also somewhat uncertain. While a quantitative assessment based on several animal studies resulted in a similar estimate of risk (which increases the confidence somewhat), the quality of the available studies was poor (that is, they were conducted at single dose levels or lacked control groups).

CARDR-

o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1986, 1991

Source Document Review -- The values in 1986 Health Assessment Document for Beryllium and the 1991 Drinking Water Criteria Document for Beryllium received Agency and external review.

Other EPA Documentation -- None
DOCUMENT

o REVIEW DATES : 05/04/88, 02/01/89, 12/07/89
o VERIFICATION DATE : 05/04/88 (inhalation); 02/01/89 (oral)
o EPA CONTACTS :

William Pepelko / OHEA -- (202)260-5904

David Bayliss / OHEA -- (202)260-5726

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA -

Considers technological or economic feasibility? -- YES

Discussion -- Beryllium was listed as a hazardous air pollutant under section 112 of the CAA in 1971 on the basis that it can cause the chronic lung disease berylliosis. Emission standards promulgated for extraction, ceramic, and propellant plants, foundries, incinerators, and machine shops are 10 g/24 hr or attainment of an ambient concentration near the source of 0.01 ug/cu.m, 30 day average. This ambient concentration was judged adequate to protect the public health with an ample margin of safety. More complex standards were also promulgated for beryllium rocket motor firing. The NESHAPs are now under review, and will consider new health evidence that beryllium may be a carcinogen. Reporting of releases of massive forms of this hazardous substance is not required if the diameter of the pieces released exceeds 100 micrometers (0.004 inches).

Reference -- 40 CFR Part 61, Subparts C & D

EPA Contact -- Emissions Standards Division, OAQPS
(917)541-5571 / FTS 629-5571

WQCHU-

Water and Fish Consumption: $6.8E-3$ ug/L

Fish Consumption Only: $1.17E-1$ ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criterion represent a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80); Quality Criteria for Water, EPA 440/5-86-001 (5/87).

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- $1.3E+2$ ug/L

Chronic LEC -- $5.3E+0$ ug/L

Marine: None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available. Hardness has a substantial effect on acute toxicity.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed MCLG for beryllium is zero based on the evidence of carcinogenic potential (B2).

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4731

MCL -

Value -- 0.001 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- YES

Discussion -- The MCL is based on 5x the MDL, which is associated with a maximum lifetime individual risk of 1 E-4.

Monitoring requirements -- Ground water systems every 3 years; surface water systems annually; will allow monitoring at up to 10-year intervals after the system completes 3 rounds of sampling at <50% of the MCL.

Analytical methodology -- Atomic absorption/furnace technique (EPA 210.2; ASTM D-3645; SM 304); inductively-coupled plasma (EPA 200.7; SM 305); ICP mass spectrometry (EPA 200.8); PQL= 0.001 mg/L.

Best available technology -- Activated alumina; ion exchange; reverse osmosis; lime softening; coagulation/filtration.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (Final, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Atomic absorption/furnace technique (EPA 210.2; SM 304; ASTM D-3645); inductively coupled plasma (EPA 200.7; SM 305); spectrophotometric (EPA 200.8).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value (status) -- 10 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for beryllium is based on potential carcinogenicity. Available data indicate a hazard ranking of medium based on a potency factor of 79.70/mg/kg/day and a weight-of-evidence group B2, which correspond to an RQ of 10 pounds. Reporting of releases of massive forms of this hazardous substance is not required if the diameter of the pieces released exceeds 100 micrometers (0.004 inches).

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Cox, G.E., D.E. Bailey and K. Morgareidge. 1975. Chronic feeding studies with beryllium sulfate in rats. Unpublished report submitted by the Food and Drug Research Laboratories, Inc., to the Aluminum Company of America, Pittsburgh, PA.

OREF - Puzanova, L., M. Daskocil and A. Doubkova. 1978. Disturbances of the development of chick embryos after the administration of beryllium chloride at early stages of embryogenesis. *Folia. Morphologica*. 26(3): 228-231.

OREF - Schroeder, H.A. and M. Mitchener. 1975. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. *J. Nutr.* 105: 421-427.

OREF - U.S. EPA. 1985. Drinking Water Criteria Document for Beryllium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

IREF - None

CREF - Bayliss, D.L. 1980. U.S. EPA, Washington, DC. Letter to William H. Foege, M.D., Center for Disease Control, Atlanta, GA. November 12.

CREF - Bayliss, D.L. and J.K. Wagoner. 1977. Bronchogenic cancer and cardio-respiratory disease mortality among white males employed in a beryllium production facility. OSHA Beryllium Hearing, 1977, Exhibit 13.F.

CREF - Cloudman, A.M., D. Vining, S. Barkulis and J.J. Nickson. 1949. Bone changes following intravenous injections of beryllium. *Am. J. Pathol.* 25: 810-811.

CREF - Gardner, L.U. and H.F. Heslington. 1946. Osteo-sarcoma from intravenous beryllium compounds in rabbits. *Fed. Proc.* 5: 221. (Cited in U.S. EPA, 1987)

CREF - Groth, D.H., C. Kommineni and G.R. Mackay. 1980. Carcinogenicity of beryllium hydroxide and alloys. *Environ. Res.* 21(1): 63-84.

CREF - Hsie, A.W., J.P. O'Neill, J.R. San Sebastian, et al. 1979. Quantitative mammalian cell genetic toxicology: Study of the cytotoxicity and mutagenicity of seventy individual environmental agents related to energy technologies and three subfractions of crude synthetic oil in the CHO/HGPRT system. *Environ. Sci. Res.* 15: 219-315.

CREF - Ishinishi, N., M. Mizunoe, T. Inamasu and A. Hisanga. 1980. Experimental study on carcinogenicity of beryllium oxide and arsenic trioxide to the lung of rats by an intratracheal instillation. *Fukuoka Igaku Zasshi*. 71(1): 19-26. (Jap. with Eng. abstract)

CREF - Larramendy, M.L., N.C. Popescu and J.A. DiPaola. 1981. Induction by inorganic metal salts of sister chromatid exchanges and chromosome aberrations in human and Syrian hamster cell strains. *Environ. Mutagen.* 3: 597-606.

CREF - MacMahon, B. 1977. Evaluation of epidemiological materials. January 10, 1978. Brush Wellman, Cleveland, OH. OSHA Beryllium Hearings: 5.

- CREF - MacMahon, B. 1978. OSHA Beryllium Hearings, comment on recent post-hearing submissions. Docket No. H005, February 9, 1979.
- CREF - Mancuso, T.F. 1970. Relation of duration of employment and prior respiratory illness to respiratory cancer among beryllium workers. *Environ. Res.* 3: 251-275.
- CREF - Mancuso, T.F. 1979. Occupational lung cancer among beryllium workers in dusts and disease. In: *Proc. Conference on Occupational Exposure to Fibrous and Particulate Dust and Their Extension into the Environment*, R. Lemen and J. Dement, Ed. Pathrotox Publishers, Inc.
- CREF - Mancuso, T.F. 1980. Mortality study of beryllium industry workers' occupational lung cancer. *Environ. Res.* 21: 48-55.
- CREF - Miyaki, M., N. Akamatsu, T. Ono, H. Koyama. 1979. Mutagenicity of metal cations in cultured cells from chinese hamster. *Mutat. Res.* 68: 259-263.
- CREF - Morgareidge, K., G.E. Cox, D.E. Bailey and M.A. Gallo. 1977. Chronic oral toxicity of beryllium in the rat. *Toxicol. Appl. Pharmacol.* 41(1): 204-205.
- CREF - Reeves, A.L., D. Deitch, and A.J. Vorwald. 1967. Beryllium carcinogenesis: I. Inhalation exposure of rats to beryllium sulfate aerosol. *Cancer Res.* 27(1): 439-445.
- CREF - Schepers, G.W.H. 1961. Neoplasia experimentally induced by beryllium compounds. *Prog. Exp. Tumor Res.* 2: 203-244.
- CREF - Schroeder, H.A. and M. Mitchener. 1975a. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. *J. Nutr.* 105: 421-427.
- CREF - Schroeder, H.A. and M. Mitchener. 1975b. Life-term effects of mercury, methyl mercury and nine other trace metals on mice. *J. Nutr.* 105: 452-458.
- CREF - Simmon, V.F., H.S. Rosenkranz, E. Zeiger and L.A. Poirier. 1979. Mutagenic activity of chemical carcinogens and related compounds in the intraperitoneal host-mediated assay. *J. Natl. Cancer Inst.* 62(4): 911-918.
- CREF - U.S. EPA. 1986. Health Assessment Document for Beryllium. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-84-026F.
- CREF - U.S. EPA. 1991. Drinking Water Criteria Document for Beryllium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.
- CREF - Vorwald, A.J. 1968. Biologic manifestations of toxic inhalants in monkeys. In: *Use of Nonhuman Primates in Drug Evaluation*, H. Vagtborg, Ed. University of Texas Press, Austin, TX. p. 222-228.
- CREF - Wagner, W.D., D.H. Groth, J.L. Holtz, G.E. Madden and H.E. Stokinger. 1969. Comparative chronic inhalation toxicity of beryllium ores, bertrandite and beryl, with production of pulmonary tumors by beryl. *Toxicol. Appl. Pharmacol.* 15: 10-29.
- CREF - Wagoner, J.K., P.F. Infante and D.L. Bayliss. 1980. Beryllium: An etiologic agent in the induction of lung cancer, nonneoplastic respiratory disease, and heart disease among industrially exposed workers. *Environ. Res.* 21: 15-34.
- HAREF- None

[IRIS] SS 4 /cf?

USER:



Attachment 6

Risk Characterization Methodologies and Results

A6-1.0 Introduction

The risk characterization evaluates and quantitatively estimates the risks associated with each of the chemicals of potential concern (COPC) in each exposure pathway for each exposure scenario, given the assumptions of the exposure assessment and toxicity criteria. Cancer risks and noncancer risks are addressed separately.

A6-2.0 Methods for Characterizing Noncancer Risks

The noncancer risk associated with a given chemical in an exposure pathway is evaluated in terms of the hazard quotient (HQ). The HQ of chemical "a" via the ingestion pathway is calculated as follows:

$$HQ_{Ing}(a) = \frac{IN_{Ing}(a)}{RfD_a}$$

If the IN_{Ing} is less than the RfD , then the HQ_{Ing} is less than a value of one and the IN_{Ing} is regarded as being unlikely to result in any adverse health effects even to the most susceptible members of a population. HQ values for the other exposure pathways are estimated similarly. The HQ does not define a particular level of risk. One reason for this is that the RfD is an estimate of a threshold exposure level, and below the threshold essentially no risk is assumed.

The sum of HQ values for the identified exposure pathways represents an estimate of the total noncancer risk associated with a given chemical, referred to as the hazard index (HI) of that chemical. The HI of chemical "a" via the ingestion, dermal absorption, and inhalation pathways is as follows:

$$HI(a) = HQ_{Ing}(a) + HQ_{Der}(a) + HQ_{Inh}(a)$$

The total hazard index (THI) represents the overall noncancer risks posed by the COPC in a given exposure scenario, and is the sum of the individual HI values:

$$THI = HI(a) + HI(b) + HI(c) + \dots + HI(n)$$

The THI is compared to a target value of 1. If the THI is less than 1, then it is unlikely, given the exposure scenario assumptions, that the COPC represent a health risk. If the THI exceeds 1, then the effects of the COPC will be broken down by target organs. If any of the target organ-specific THI values exceed 1, then a potential for adverse health effects may be indicated. If all target organ-specific THI values are less than 1, then adverse noncarcinogenic health effects are not considered likely.

A6-3.0 Methods for Characterizing Cancer Risks

With regard to carcinogenic effects, the calculated cancer risk of a given compound in an exposure pathway is simply referred to as the cancer risk (CR). The CR of chemical "a" via the ingestion pathway is calculated as follows:

$$CR_{Ing}(a) = 1 - e^{(-CSF(a) \times IN_{Ing})}$$

The CR for other exposure pathways are estimated similarly. The cancer risk of a given compound, considering all exposure pathways, is referred to here as the chemical cancer risk (CCR) and is calculated as follows:

$$CCR(a) = CR_{Ing}(a) + CR_{Der}(a) + CR_{Inh}(a)$$

The estimated incremental lifetime cancer risk (ILCR) represents the overall risks posed by all COPC in a given exposure scenario, and is the sum of all the CCR values:

$$ILCR = CCR(a) + CCR(b) + \dots + CCR(n)$$

The ILCR is compared to a target risk range that is considered protective of human health, generally between 10^{-6} and 10^{-4} .

A6-4.0 Risk Characterization Results

Risk characterization calculations and results for each Site area are shown in Tables A6-1 through A6-4 of this attachment. Included on these tables are the exposure results from Attachment 3 of the Risk Assessment and toxicity criteria summarized from Section A5.0 of the Risk Assessment text.

TABLE A6-1
PRODUCTION AREA
ON-SITE WORKER SCENARIO
NONCANCER RISK CALCULATIONS

CHEMICAL	INTAKE				RfD _o (a)	RfD _i (b)	HAZARD QUOTIENT				HAZARD INDEX
	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhal. - FDE (mg/kg-day)	Inhal. - VE (mg/kg-day)			Ingestion	Dermal	Inhal. - FDE	Inhal. - VE	
PCB 1248 - Dev. (c)	6.89E-08	1.03E-07	6.00E-11	1.22E-09	8.00E-05(d)	8.00E-05(e)	8.61E-04	1.28E-03	7.50E-07	1.53E-05	0.0022
PCB 1248 - Imm. (f)	6.89E-08	1.03E-07	6.00E-11	1.22E-09	1.00E-03(g)	1.00E-03(e)	6.89E-05	1.03E-04	6.00E-08	1.22E-06	0.0002
PCB 1254	5.64E-07	8.39E-07	4.91E-10	2.89E-09	2.00E-05	2.00E-05(e)	2.82E-02	4.19E-02	2.45E-05	1.44E-04	0.0703
COMBINED PCB TOTAL HAZARD INDEX (h)											0.0705

- a. Chronic reference dose, oral exposure route. Source: Integrated Risk Information System database (IRIS), unless otherwise noted.
- b. Chronic reference dose, inhalation exposure route. Calculated from reference concentrations (RfCs) as listed on IRIS, unless otherwise noted.
- c. HI is based on developmental effects; not additive with the HI for PCB 1254.
- d. Provisional RfD_o derived based on developmental effects in Rhesus monkeys. Refer to Section A6.2.1 of the Risk Assessment.
- e. No RfD_i or reference concentration (RfC) exists; the value used as the RfD_o was used as a provisional RfD_i.
- f. HI is based on immunologic effects; may be added with the HI of PCB 1254.
- g. Provisional RfD_o based on immunologic effects in Rhesus monkeys. Refer to Section A6.2.1 of the Risk Assessment.
- h. The sum of the HI values for PCB 1254 and the immunologic effects HI value of PCB 1248.

TABLE A6-2
PRODUCTION AREA
ON-SITE WORKER SCENARIO
CANCER RISKS

CHEMICAL	INTAKE				CSF _o (a)	CSF _i (b)	CANCER RISK				
	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhal. - FDE (mg/kg-day)	Inhal. - VE (mg/kg-day)			Ingestion	Dermal	Inhal. - FDE	Inhal. - VE	Combined Routes
PCB 1260	3.18E-07	4.74E-07	2.78E-10	1.88E-09	7.70E+00	7.70E+00	2.45E-06	3.65E-06	2.14E-09	1.45E-08	6.12E-06
<i>gamma</i> -Chlordane	6.78E-09	1.98E-08	5.93E-12	2.29E-10	1.30E+00	1.30E+00	8.82E-09	2.57E-08	7.70E-12	2.98E-10	3.49E-08
Total PCBs as 1260 (c)	3.08E-07	4.58E-07	2.69E-10	3.25E-09	7.70E+00(d)	7.70E+00	2.37E-06	3.53E-06	2.07E-09	2.50E-08	5.93E-06
INCREMENTAL LIFETIME CANCER RISK (e)											6.15E-06

- a. Cancer slope factor, oral exposure route. Source: Integrated Risk Information System database (IRIS), unless otherwise noted.
- b. Cancer slope factor, inhalation exposure route. No CSF_is were available for these compounds; the CSF_o values were substituted.
- c. In accordance with Region 1 policy, risk of total PCBs was calculated assuming all PCBs have the same cancer potency as PCB 1260. This policy is not consistent with toxicological data which indicate that PCB 1248 and PCB 1254 are not carcinogens.
- d. Assumed to be the same as for PCB 1260 (see footnote d).
- e. Includes the sum of the estimated potential cancer risks associated with PCB 1260 and *gamma*-chlordane.

TABLE A6-3
WARWICK AREA
ON-SITE RESIDENTIAL SCENARIO
NONCANCER RISK CALCULATIONS

CHEMICAL	INTAKE				RfD _o (a)	RfD _i (b)	HAZARD QUOTIENT				HAZARD INDEX
	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation-FD (mg/kg-day)	Inhalation-VE (mg/kg-day)			Ingestion	Dermal	Inhalation FD	Inhalation VE	
PCB 1248 - Dev. (c)	2.52E-05	8.38E-06	8.90E-09	2.45E-07	8.00E-05(d)	8.00E-05(e)	3.15E-01	1.05E-01	1.11E-04	3.06E-03	0.423
PCB 1248 - Imm. (f)	2.52E-05	8.38E-06	8.90E-09	2.45E-07	1.00E-03(g)	1.00E-03(e)	2.52E-02	8.38E-03	8.90E-06	2.45E-04	0.034
PCB 1254	8.74E-06	2.90E-06	3.08E-09	2.07E-08	2.00E-05(d)	2.00E-05(c)	4.37E-01	1.45E-01	1.54E-04	1.03E-03	0.583
2-Nitroaniline	1.18E-05	7.24E-06	4.15E-09	2.47E-07	5.71E-05(h)	5.71E-05(i)	2.06E-01	1.27E-01	7.26E-05	4.32E-03	0.337
Methoxychlor	3.90E-04	2.54E-04	1.37E-07	0.00E+00	5.00E-03	5.00E-03(e)	7.80E-02	5.08E-02	2.74E-05	0.00E+00	0.129
COMBINED PCBs TOTAL HAZARD INDEX (j)											0.617

- a. Chronic reference dose, oral exposure. Source: Integrated Risk Information System database (IRIS), unless otherwise noted.
- b. Chronic reference dose, inhalation exposure.
- c. HI is based on developmental effects.
- d. Provisional RfD_o derived based on developmental effects in Rhesus monkeys. Refer to Section A6.2.1 of the Risk Assessment.
- e. No RfD_i or reference concentration (RfC) exists; the value used also as the RfD_o was used as a provisional RfD_i.
- f. HI is based on immunologic effects; may be added with the HI of PCB 1254.
- g. Provisional RfD_o based on immunologic effects in Rhesus monkeys. Refer to Section A6.2.1 of the Risk Assessment.
- h. No RfD_o exists; the RfD_i was used as a provisional RfD_o.
- i. Derived from the RfC of 2×10^{-4} mg/m³. Source of RfC: HEAST.
- j. The sum of the HI values for PCB 1254 and the immunologic effects HI value of PCB 1248.

TABLE A6-4
WARWICK AREA
ON-SITE RESIDENTIAL SCENARIO
CANCER RISK CALCULATIONS

CHEMICAL	INTAKE				CSF _o (a) (mg/kg-day) ⁻¹	CSF _i (b) (mg/kg-day) ⁻¹	CANCER RISK				
	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation-FD (mg/kg-day)	Inhalation-VE (mg/kg-day)			Ingestion	Dermal	Inhalation FD	Inhalation VE	Combined Routes
Aldrin	1.41E-07	9.20E-08	4.98E-11	2.49E-09	1.70E+01	1.70E+01	2.40E-06	1.56E-06	8.47E-10	4.23E-08	4.01E-06
Beryllium	4.84E-07	2.68E-06	1.71E-10	0.00E+00	4.30E+00	8.40E+00(c)	2.08E-06	1.15E-05	1.43E-09	0.00E+00	1.36E-05
Dieldrin	1.08E-07	7.01E-08	3.80E-11	2.21E-09	1.60E+01	1.60E+01	1.72E-06	1.12E-06	6.08E-10	3.54E-08	2.88E-06
Heptachlor epoxide	1.28E-07	8.32E-08	4.51E-11	1.21E-08	9.10E+00	9.10E+00	1.16E-06	7.57E-07	4.10E-10	1.10E-07	2.03E-06
Total PCBs as 1260 (d)	1.21E-05	4.02E-06	4.32E-09	1.06E-07	7.70E+00(e)	7.70E+00(f)	9.32E-05	3.10E-05	3.32E-08	8.16E-07	1.25E-04
INCREMENTAL LIFETIME CANCER RISK											1.47E-04

a. Cancer slope factor, oral route. Source: Integrated Risk Information System database (IRIS), unless otherwise noted.

b. Cancer slope factor, inhalation route.

c. Derived from an inhalation unit risk of 2.4E-03 (ug/m³)⁻¹. Source: IRIS.

d. In accordance with Region 1 policy, risk of total PCBs was calculated assuming all PCBs have the same cancer potency as PCB 1260. This policy is not consistent with toxicological data which indicate that PCB 1248 and PCB 1254 are not carcinogens.

e. Assumed to be the same as for PCB 1260 (see footnote "e").

f. No CSF_i is available; the CSF_o is used as a provisional CSF_i.

PCB LABORATORY INFORMATION AND METHODOLOGY

Soil samples were analyzed by three environmental laboratories: Radian, Inc., Savannah Laboratories, and CIBA-GEIGY Corporation Environmental Testing Laboratory (CETL). Both Radian and Savannah analyzed the soil samples for Appendix IX PCBs. CETC analyzed the soil samples for PCBs using engineering grade methods.

Prior to analyzing the soil samples, each of the laboratories was required to submit a Quality Assurance Project Plan (QAPP) to USEPA Region I for review and comment. Each of the plans was approved by the USEPA. All of the plans are contained in the RCRA Facility Investigation Quality Assurance Documents: Supplement dated January 1992.

For this soil investigation, Radian and Savannah Laboratories used Method 8080 (EPA Document SW 846 Test Methods for Evaluation of Solid Waste Physical/Chemical Methods). Soil samples analyzed by CETL using engineering grade methods were for supplemental information and were not considered data for risk based determinations.

FIELD QUALITY ASSURANCE/QUALITY CONTROL

Field blanks were analyzed to check for cross-contamination from field equipment. Field blanks were collected at the rate of one per 20 samples, and were analyzed for the same parameters as the associated samples. Field blanks were made by pouring laboratory-supplied distilled deionized water over the sampling equipment and into laboratory sample containers.

Field duplicates were collected to check the reproducibility of laboratory data by comparing analytical results for two samples from the same location. Field duplicates were collected at the rate of one per 20 samples, and were analyzed for the same parameters as the associated samples.

DATA VALIDATION

The laboratory hardcopy deliverables were submitted to WCC for validation. Data were evaluated using the following quality control criteria:

- data completeness;
- sample holding times;
- calibrations;
- blank results;
- surrogate recoveries;
- matrix spike/matrix spike duplicate results;
- field duplicate results;
- pesticide instrument performance; and,
- compound quantification.

Data Completeness

For purposes of this data validation, data packages were considered complete if the packages contained the list above, plus laboratory case narratives and chain-of-custody information.

Sample Holding Times

Sample holding times for soils were 14 days to extraction and 40 days to analyses. If holding times were exceeded, all positive hits were estimated (qualified J) and all negative results were estimated (qualified UJ). If holding times were grossly exceeded, the reviewer may determine that non-detects were also unusable (qualified I).

Calibrations

Calibrations were reviewed as a measure of the laboratory's accuracy. For the initial pesticide/PCB calibration, all compounds were required to meet the percent Relative Standard Deviation (RSD) of less than 20% for the initial calibration on the quantification column. Failure to meet this requirement resulted in the estimation of

positive results (qualified J).

For the pesticide/PCB continuing calibration, all compounds were required to meet the percent difference (%D) criteria of $\pm 15\%$ on the quantification column and $\pm 20\%$ on the confirmation column. If the %D criteria is not achieved, all positive results were estimated by the data reviewer (qualified J).

Blank Results

Any positive results for Appendix IX compounds present in the blank require the reviewer to qualify positive results in the associated samples. Any results that were qualified due to blank contamination were listed in the data validation narrative.

Surrogate Recoveries

The surrogate recovery range for dibutylchlorodate in soils is 20-150%. Non-detected results were rejected (R) if the surrogate recovery was less than 10% and estimated (J) if the surrogate recovery was greater than 0% but less than 20%. Positive results were estimated J) if the surrogate recovery was outside of the 20-150% range. Surrogate recovery is a measure of the laboratory's accuracy.

Matrix Spike/Matrix Spike Duplicate Results

Matrix spike and matrix spike duplicate sample results were estimated (J) if the established relative percent difference (RPD) criteria were not achieved. RPD criteria for matrix spike/matrix spike duplicates are a measure of the laboratory's precision and accuracy.

Field Duplicate Results

Field duplicate sample results were estimated (UJ, J) if the RPD criteria was not achieved (50%).

Pesticide Instrument Performance

Compounds were required to date within pre-established retention time windows to meet the compound identification criteria. Instrument performance was also judged by DDT minimum retention times and DDT/endrin breakdown results; however, these results were required for data completeness; effects on reported PCB results are treated on a case by case basis. As another control, the dibutylchlorendate retention time shifts were measured for all analyses. If the retention time shifts were outside 1.5%, the data was qualified or rejected based on the professional judgment of the reviewer.

Compound Identification/Quantification

Positive detects for PCBs were confirmed on a second column with a different stationary phase. Also, quantification algorithms were periodically checked by the data reviewer. The results from both analytical columns must indicate reasonable agreement between the analyses. Justification of sample results was based upon the professional judgment of the reviewer.

SUMMARY OF PCB DATA IN THE PRODUCTION AREA (AOC-13)

During the RFI, 142 soil samples were collected and analyzed for Appendix IX PCBs in the Production Area. These data were validated by WCC using the protocols presented in the data validation checklist - Organic Analyses Laboratory Data Review USEPA Region I Worksheets, Edited for Appendix IX Compounds, Revision I, January 1992.

This checklist was prepared with USEPA Region I guidance. It was submitted as part of the RCRA Facility Investigation Quality Assurance Documents: Supplement dated January 1992 and was subsequently approved by USEPA Region I. In addition, 18 soil samples were analyzed for PCBs using engineering-grade methods. These data were used to help delineate the extent of contamination. Engineering-grade PCB data were not validated or used in the risk assessment conducted by PTRL. Only the Appendix IX data that passed validation were used in the risk assessment.

Approximately 13% of the overall data were rejected as a result of validation. Rejection of data for Aroclors 1016, 1221, 1232, 1242 and 1260 averaged 14.1%. Rejection of data for Aroclors 1248 and 1254 averaged 1.76%. Rejection of data for Aroclors 1016, 1221, 1232, 1242, 1260 was higher than rejected data for Aroclors 1248 and 1254 because Aroclors 1248 and 1254 were the only detected Aroclors, except for Arochlor 1260 which was detected in some samples. The difference is explained by validation guidelines which required rejection of non-detected results and qualification of detected results as estimated.

A statistical summary of the detection limits for all the Production Area PCB data is presented below. For each Aroclor, the minimum, maximum, and average detection limits are presented (along with the variance, standard deviation, and 10th and 90th percentiles). All of the data are given in ppm.

Statistical Summary of Detection Limits for Production Area PCB Data

Aroclor	Min.	Max.	Avg.	Var.	Stdev.	10th Percentile	90th Percentile
1016	400	3.67	1310	36.2	0.034	0.975	984
1221	0.02	820	7.46	5506	74.2	0.069	1.95
1232	0.02	400	3.83	1310	36.2	0.034	1.91
1242	0.01	400	3.66	1310	36.2	0.034	0.975
1248	0.01	2.3	0.29	0.189	0.44	0.034	1
1254	0.02	400	22.2	8379	91.54	0.0322	4.48
1260	0.02	400	4.26	1466	38.28	0.034	2.1

Detected results for all Production Area PCB data ranged from 0.02 ppm to 430 ppm. The highest concentrations were detected for Aroclor 1248. The ranges of detected results for each Aroclor are presented below.

Production Area PCB Data

Aroclor	Frequency Detected	Minimum Detected	Maximum Detected
1016	0	-	-
1221	0	-	-
1232	0	-	-
1242	0	-	-
1248	37	0.02	4500
1254	122	0.05	84
1260	14	0.07	13

SUMMARY OF PCB DATA IN THE WARWICK AREA (SWMU-5)

During the RFI, there were 29 soil samples collected and analyzed for Appendix IX PCBs in the Warwick Area. The data were validated by WCC using the protocols in the data validation checklist - Organic Analyses Laboratory Data Review - USEPA Region I Worksheets, Edited for Appendix IX Compounds, Revised January 1, 1992. Only the Appendix IX data that passed validation were used in the risk assessment that was conducted by PTRL.

Approximately 9% of the overall data were rejected as a result of validation. Rejection of data for Aroclors 1016, 1221, 1232, 1242, 1254 and 1260 averaged 10.3%. Rejection of data for Aroclor 1248 was 3.4%.

A statistical summary of the detection limits for all the SWMU-5 PCB data is presented below. For each Aroclor, the minimum, maximum, and average detection limits are presented (along with the variance, standard deviation, and 10th and 90th percentiles). All data are given in ppm.

Statistical Summary of Detection Limits for SWMU-5 PCB Data

Aroclor	Min.	Max	Avg.	Var.	Stdev.	10th Percentile	90th Percentile
1016	0.011	25	1.83	25.5	5.05	0.023	3.5
1221	0.021	50	3.64	101	10.09	0.047	7
1232	0.021	50	3.03	95.7	9.78	0.029	4.75
1242	0.011	25	1.83	25.5	5.05	0.023	3.5
1248	0.011	25	1.83	26.5	5.15	0.02	3.68
1254	0.023	50	1.89	152	12.36	0.035	6.6
1260	0.021	50	4.54	95.7	9.78	0.029	4.75

Detected results ranged from 0.073 ppm to 160 ppm for all Aroclors. Detected results for each Aroclor are listed below:

SWMU-5 PCB Data

Aroclor	Frequency Detected	Minimum Detected	Maximum Detected
1016	0	-	-
1221	0	-	-
1232	0	-	-
1242	0	-	-
1248	3	8.1	160
1254	10	0.073	36
1260	0	-	-

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Report To: Michael Goodman
Address: Ciba-Geigy Corp.
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Project Name: TCLP Characterization

Charge Number: CLA-61

Report Number: 94J-1005

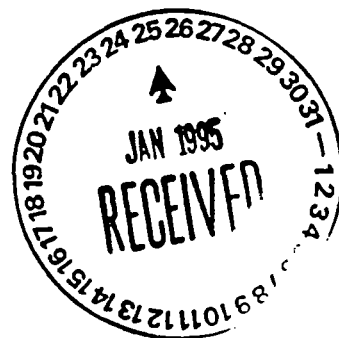
Report Date: 01-24-1995

NJ Laboratory ID#: 15265



Dr. David R. Ellis
Laboratory Manager

C:



EXTRACTION DATE LOG

Sample #	Sample Description	Parameter	Date Extracted
94120934	TCLP-N45	TCLP - All Parameters	1/02/1995
		Semivolatiles - TCLP	1/04/1995
		Pesticides - TCLP	1/04/1995
		Herbicides - TCLP	1/06/1995
94120935	TCLP-O44	TCLP - All Parameters	1/02/1995
		Semivolatiles - TCLP	1/04/1995
		Pesticides - TCLP	1/04/1995
		Herbicides - TCLP	1/06/1995
94120936	TCLP-P43	TCLP - All Parameters	1/02/1995
		Semivolatiles - TCLP	1/04/1995
		Pesticides - TCLP	1/04/1995
		Herbicides - TCLP	1/06/1995
94120937	TCLP-C3	TCLP - All Parameters	1/02/1995
		Semivolatiles - TCLP	1/04/1995
		Pesticides - TCLP	1/04/1995
		Herbicides - TCLP	1/06/1995
94120938	TCLP-D3	TCLP - All Parameters	1/02/1995
		Semivolatiles - TCLP	1/04/1995
		Pesticides - TCLP	1/04/1995
		Herbicides - TCLP	1/06/1995
94120939	TCLP-B5G	TCLP - All Parameters	1/02/1995
		Semivolatiles - TCLP	1/04/1995
		Pesticides - TCLP	1/04/1995
		Herbicides - TCLP	1/09/1995
94120940	TCLP-C2	TCLP - All Parameters	1/02/1995
		Semivolatiles - TCLP	1/04/1995
		Pesticides - TCLP	1/04/1995
		Herbicides - TCLP	1/09/1995
94120941	TCLP-B5C	TCLP - All Parameters	1/02/1995
		Semivolatiles - TCLP	1/04/1995
		Pesticides - TCLP	1/04/1995
		Herbicides - TCLP	1/09/1995
94120942	TCLP-ZZ3	TCLP - All Parameters	1/02/1995
		Semivolatiles - TCLP	1/04/1995
		Pesticides - TCLP	1/04/1995
		Herbicides - TCLP	1/09/1995
94120943	TCLP-D2	TCLP - All Parameters	1/02/1995
		Semivolatiles - TCLP	1/04/1995
		Pesticides - TCLP	1/04/1995
		Herbicides - TCLP	1/09/1995
95010024	TCLPMS SA# 94120943	Semivolatiles	1/04/1995
		Pesticides	1/04/1995
		Herbicides	1/09/1995

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TCLP REGULATORY LEVELS

<u>PARAMETER</u>	<u>MAXIMUM</u> <u>CONC.</u> <u>LIMITS</u>
Vinyl chloride	200
1,1-Dichloroethene	700
2-Butanone	200000
Chloroform	6000
Carbon tetrachloride	500
1,2 Dichloroethene	500
Trichloroethene	500
Benzene	500
Tetrachloroethene	700
Chlorobenzene	100000
1,4-Dichlorobenzene	7500
Total Cresols	200000
Hexachloroethane	3000
Nitrobenzene	2000
Hexachlorobutadiene	500
2,4,6-Trichlorophenol	2000
2,4,5-Trichlorophenol	400000
2,4-Dinitrotoluene	130
Hexachlorobenzene	130
Pentachlorophenol	100000
Pyridine	5000
Gamma BHC (Lindane)	400
Chlordane	30
Endrin	20
Heptachlor	8.0
Heptachlor Epoxide	8.0
Toxaphene	500
Methoxychlor	10000
2,4-D	10000
2,4,5-TP(Silvex)	1000
Barium	100000
Cadmium	1000
Chromium	5000
Arsenic	5000
Lead	5000
Selenium	1000
Silver	5000
Mercury	200
	(ug/L)

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-N45
ETL Sample # : 94120934
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/04/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
Vinyl chloride	ND	100
1,1-Dichloroethene	ND	50
2-Butanone	ND	1000
Chloroform	ND	50
Carbon tetrachloride	ND	50
1,2-Dichloroethane	ND	50
Trichloroethene	ND	50
Benzene	ND	50
Tetrachloroethene	ND	50
Chlorobenzene	ND	50
##1,2-Dichloroethane-d4	107 %	76 to 114
##Toluene-d8	100 %	88 to 110
##4-Bromofluorobenzene	103 %	86 to 115

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

J Indicates detected but below method detection limits

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-044
ETL Sample # : 94120935
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/05/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
Vinyl chloride	ND	100
1,1-Dichloroethene	ND	50
2-Butanone	ND	1000
Chloroform	ND	50
Carbon tetrachloride	ND	50
1,2-Dichloroethane	ND	50
Trichloroethene	ND	50
Benzene	ND	50
Tetrachloroethene	ND	50
Chlorobenzene	ND	50
##1,2-Dichloroethane-d4	109 %	76 to 114
##Toluene-d8	101 %	88 to 110
##4-Bromofluorobenzene	104 %	86 to 115

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration equal to or greater than 10% of the analyte concentration in the sample
MDL Method Detection Limit

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-P43
ETL Sample # : 94120936
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/05/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
Vinyl chloride	ND	100
1,1-Dichloroethene	ND	50
2-Butanone	ND	1000
Chloroform	ND	50
Carbon tetrachloride	ND	50
1,2-Dichloroethane	ND	50
Trichloroethene	ND	50
Benzene	ND	50
Tetrachloroethene	ND	50
Chlorobenzene	ND	50
##1,2-Dichloroethane-d4	108 %	76 to 114
##Toluene-d8	101 %	88 to 110
##4-Bromofluorobenzene	104 %	86 to 115

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

J Indicates detected but below method detection limits

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-C3
ETL Sample # : 94120937
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/05/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
Vinyl chloride	ND	100
1,1-Dichloroethene	ND	50
2-Butanone	ND	1000
Chloroform	ND	50
Carbon tetrachloride	ND	50
1,2-Dichloroethane	ND	50
Trichloroethene	ND	50
Benzene	ND	50
Tetrachloroethene	ND	50
Chlorobenzene	ND	50
##1,2-Dichloroethane-d4	106 %	76 to 114
##Toluene-d8	101 %	88 to 110
##4-Bromofluorobenzene	101 %	86 to 115

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

J Indicates detected but below method detection limits

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-D3
ETL Sample # : 94120938
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/05/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
Vinyl chloride	ND	100
1,1-Dichloroethene	ND	50
2-Butanone	ND	1000
Chloroform	ND	50
Carbon tetrachloride	ND	50
1,2-Dichloroethane	ND	50
Trichloroethene	ND	50
Benzene	ND	50
Tetrachloroethene	ND	50
Chlorobenzene	ND	50
##1,2-Dichloroethane-d4	108 %	76 to 114
##Toluene-d8	103 %	88 to 110
##4-Bromofluorobenzene	103 %	86 to 115

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

J Indicates detected but below method detection limits

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

REPORT NUMBER: 94J-1005

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VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-B5G
ETL Sample # : 94120939
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/05/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
Vinyl chloride	ND	100
1,1-Dichloroethene	ND	50
2-Butanone	ND	1000
Chloroform	ND	50
Carbon tetrachloride	ND	50
1,2-Dichloroethane	ND	50
Trichloroethene	ND	50
Benzene	ND	50
Tetrachloroethene	ND	50
Chlorobenzene	ND	50
##1,2-Dichloroethane-d4	106 %	76 to 114
##Toluene-d8	101 %	88 to 110
##4-Bromofluorobenzene	102 %	86 to 115

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-C2
ETL Sample # : 94120940
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/06/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
Vinyl chloride	ND	100
1,1-Dichloroethene	ND	50
2-Butanone	ND	1000
Chloroform	ND	50
Carbon tetrachloride	ND	50
1,2-Dichloroethane	ND	50
Trichloroethene	ND	50
Benzene	ND	50
Tetrachloroethene	ND	50
Chlorobenzene	ND	50
##1,2-Dichloroethane-d4	108 %	76 to 114
##Toluene-d8	101 %	88 to 110
##4-Bromofluorobenzene	104 %	86 to 115

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-B5C
ETL Sample # : 94120941
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/06/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
Vinyl chloride	ND	100
1,1-Dichloroethene	ND	50
2-Butanone	ND	1000
Chloroform	ND	50
Carbon tetrachloride	ND	50
1,2-Dichloroethane	ND	50
Trichloroethene	ND	50
Benzene	ND	50
Tetrachloroethene	ND	50
Chlorobenzene	ND	50
##1,2-Dichloroethane-d4	101 %	76 to 114
##Toluene-d8	102 %	88 to 110
##4-Bromofluorobenzene	97 %	86 to 115

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

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VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-ZZ3
ETL Sample # : 94120942
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/06/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
Vinyl chloride	ND	100
1,1-Dichloroethene	ND	50
2-Butanone	ND	1000
Chloroform	ND	50
Carbon tetrachloride	ND	50
1,2-Dichloroethane	ND	50
Trichloroethene	ND	50
Benzene	ND	50
Tetrachloroethene	ND	50
Chlorobenzene	ND	50
##1,2-Dichloroethane-d4	98 %	76 to 114
##Toluene-d8	100 %	88 to 110
##4-Bromofluorobenzene	101 %	86 to 115

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration equal to or greater than 10% of the analyte concentration in the sample
MDL Method Detection Limit

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-D2
ETL Sample # : 94120943
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/06/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
Vinyl chloride	ND	100
1,1-Dichloroethene	ND	50
2-Butanone	ND	1000
Chloroform	ND	50
Carbon tetrachloride	ND	50
1,2-Dichloroethane	ND	50
Trichloroethene	ND	50
Benzene	ND	50
Tetrachloroethene	ND	50
Chlorobenzene	ND	50
##1,2-Dichloroethane-d4	100 %	76 to 114
##Toluene-d8	103 %	88 to 110
##4-Bromofluorobenzene	101 %	86 to 115

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: METHOD BLANK
ETL Sample # : 95010100
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 1/04/1995
Date Received:
Date Analyzed: 1/04/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
Vinyl chloride	ND	10
1,1-Dichloroethene	ND	5.0
2-Butanone	ND	100
Chloroform	ND	5.0
Carbon tetrachloride	ND	5.0
1,2-Dichloroethane	ND	5.0
Trichloroethene	ND	5.0
Benzene	ND	5.0
Tetrachloroethene	ND	5.0
Chlorobenzene	ND	5.0
##1,2-Dichloroethane-d4	99 %	76 to 114
##Toluene-d8	100 %	88 to 110
##4-Bromofluorobenzene	101 %	86 to 115

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
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VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLPMS SA# 94120943
ETL Sample # : 95010024
Concentration Units: %
Method: 8240X

Date Sampled : 1/03/1995
Date Received: 1/03/1995
Date Analyzed: 1/05/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
Volatiles - TCLPMS	18	N/A

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLPMS SA# 94120943
ETL Sample # : 95010024
Concentration Units: %
Method: 8240

Date Sampled : 1/03/1995
Date Received: 1/03/1995
Date Analyzed: 1/05/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
##1,2-Dichloroethane-d4	108 %	76 to 114
##Toluene-d8	102 %	88 to 110
##4-Bromofluorobenzene	105 %	86 to 115

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: METHOD BLANK
ETL Sample # : 95010131
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 1/05/1995
Date Received:
Date Analyzed: 1/05/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
Vinyl chloride	ND	10
1,1-Dichloroethene	ND	5.0
2-Butanone	ND	100
Chloroform	ND	5.0
Carbon tetrachloride	ND	5.0
1,2-Dichloroethane	ND	5.0
Trichloroethene	ND	5.0
Benzene	ND	5.0
Tetrachloroethene	ND	5.0
Chlorobenzene	ND	5.0
##1,2-Dichloroethane-d4	104 %	76 to 114
##Toluene-d8	101 %	88 to 110
##4-Bromofluorobenzene	101 %	86 to 115

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

VOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: METHOD BLANK
ETL Sample # : 95010183
Concentration Units: ug/L
Method: 8240TCLP

Date Sampled : 1/06/1995
Date Received:
Date Analyzed: 1/06/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
Vinyl chloride	ND	10
1,1-Dichloroethene	ND	5.0
2-Butanone	ND	100
Chloroform	ND	5.0
Carbon tetrachloride	ND	5.0
1,2-Dichloroethane	ND	5.0
Trichloroethene	ND	5.0
Benzene	ND	5.0
Tetrachloroethene	ND	5.0
Chlorobenzene	ND	5.0
##1,2-Dichloroethane-d4	96 %	76 to 114
##Toluene-d8	101 %	88 to 110
##4-Bromofluorobenzene	99 %	86 to 115

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

J Indicates detected but below method detection limits

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-N45
ETL Sample # : 94120934
Concentration Units: ug/L
Method: 8270TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/04/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
1,4-Dichlorobenzene	ND	40
Total Cresol	ND	40
Hexachloroethane	ND	40
Nitrobenzene	ND	40
Hexachlorobutadiene	ND	40
2,4,6-Trichlorophenol	ND	40
2,4,5-Trichlorophenol	ND	40
2,4-Dinitrotoluene	ND	40
Hexachlorobenzene	ND	40
Pentachlorophenol	ND	200
Pyridine	ND	80
##2-Fluorophenol	43 %	21 to 100
##Phenol-d6	52 %	10 to 94
##Nitrobenzene-d5	66 %	35 to 114
##2-Fluorobiphenyl	59 %	43 to 116
##2,4,6-Tribromophenol	82 %	10 to 123
##Terphenyl-d14	67 %	33 to 141

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-044
ETL Sample # : 94120935
Concentration Units: ug/L
Method: 8270TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/04/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
1,4-Dichlorobenzene	ND	40
Total Cresol	ND	40
Hexachloroethane	ND	40
Nitrobenzene	ND	40
Hexachlorobutadiene	ND	40
2,4,6-Trichlorophenol	ND	40
2,4,5-Trichlorophenol	ND	40
2,4-Dinitrotoluene	ND	40
Hexachlorobenzene	ND	40
Pentachlorophenol	ND	200
Pyridine	ND	80
##2-Fluorophenol	53 %	21 to 100
##Phenol-d6	60 %	10 to 94
##Nitrobenzene-d5	67 %	35 to 114
##2-Fluorobiphenyl	60 %	43 to 116
##2,4,6-Tribromophenol	87 %	10 to 123
##Terphenyl-d14	67 %	33 to 141

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

J Indicates detected but below method detection limits

B Indicates analyte detected in blank at a concentration equal to or greater than 10% of the analyte concentration in the sample

MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-P43
ETL Sample # : 94120936
Concentration Units: ug/L
Method: 8270TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/04/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
1,4-Dichlorobenzene	ND	40
Total Cresol	ND	40
Hexachloroethane	ND	40
Nitrobenzene	ND	40
Hexachlorobutadiene	ND	40
2,4,6-Trichlorophenol	ND	40
2,4,5-Trichlorophenol	ND	40
2,4-Dinitrotoluene	ND	40
Hexachlorobenzene	ND	40
Pentachlorophenol	ND	200
Pyridine	ND	80
##2-Fluorophenol	63 %	21 to 100
##Phenol-d6	67 %	10 to 94
##Nitrobenzene-d5	68 %	35 to 114
##2-Fluorobiphenyl	63 %	43 to 116
##2,4,6-Tribromophenol	89 %	10 to 123
##Terphenyl-d14	69 %	33 to 141

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-C3
ETL Sample # : 94120937
Concentration Units: ug/L
Method: 8270TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/04/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
1,4-Dichlorobenzene	ND	40
Total Cresol	ND	40
Hexachloroethane	ND	40
Nitrobenzene	ND	40
Hexachlorobutadiene	ND	40
2,4,6-Trichlorophenol	ND	40
2,4,5-Trichlorophenol	ND	40
2,4-Dinitrotoluene	ND	40
Hexachlorobenzene	ND	40
Pentachlorophenol	ND	200
Pyridine	ND	80
##2-Fluorophenol	59 %	21 to 100
##Phenol-d6	63 %	10 to 94
##Nitrobenzene-d5	70 %	35 to 114
##2-Fluorobiphenyl	64 %	43 to 116
##2,4,6-Tribromophenol	87 %	10 to 123
##Terphenyl-d14	70 %	33 to 141

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

J Indicates detected but below method detection limits

B Indicates analyte detected in blank at a concentration equal to or greater than 10% of the analyte concentration in the sample

MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-D3
ETL Sample # : 94120938
Concentration Units: ug/L
Method: 8270TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/04/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
1,4-Dichlorobenzene	ND	40
Total Cresol	ND	40
Hexachloroethane	ND	40
Nitrobenzene	ND	40
Hexachlorobutadiene	ND	40
2,4,6-Trichlorophenol	ND	40
2,4,5-Trichlorophenol	ND	40
2,4-Dinitrotoluene	ND	40
Hexachlorobenzene	ND	40
Pentachlorophenol	ND	200
Pyridine	ND	80
##2-Fluorophenol	65 %	21 to 100
##Phenol-d6	68 %	10 to 94
##Nitrobenzene-d5	73 %	35 to 114
##2-Fluorobiphenyl	70 %	43 to 116
##2,4,6-Tribromophenol	90 %	10 to 123
##Terphenyl-d14	69 %	33 to 141

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-B5G
ETL Sample # : 94120939
Concentration Units: ug/L
Method: 8270TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/04/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
1,4-Dichlorobenzene	ND	40
Total Cresol	ND	40
Hexachloroethane	ND	40
Nitrobenzene	ND	40
Hexachlorobutadiene	ND	40
2,4,6-Trichlorophenol	ND	40
2,4,5-Trichlorophenol	ND	40
2,4-Dinitrotoluene	ND	40
Hexachlorobenzene	ND	40
Pentachlorophenol	ND	200
Pyridine	ND	80
##2-Fluorophenol	59 %	21 to 100
##Phenol-d6	65 %	10 to 94
##Nitrobenzene-d5	65 %	35 to 114
##2-Fluorobiphenyl	62 %	43 to 116
##2,4,6-Tribromophenol	84 %	10 to 123
##Terphenyl-d14	71 %	33 to 141

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-C2
ETL Sample # : 94120940
Concentration Units: ug/L
Method: 8270TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/04/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
1,4-Dichlorobenzene	ND	40
Total Cresol	ND	40
Hexachloroethane	ND	40
Nitrobenzene	ND	40
Hexachlorobutadiene	ND	40
2,4,6-Trichlorophenol	ND	40
2,4,5-Trichlorophenol	ND	40
2,4-Dinitrotoluene	ND	40
Hexachlorobenzene	ND	40
Pentachlorophenol	ND	200
Pyridine	ND	80
##2-Fluorophenol	47 %	21 to 100
##Phenol-d6	55 %	10 to 94
##Nitrobenzene-d5	70 %	35 to 114
##2-Fluorobiphenyl	68 %	43 to 116
##2,4,6-Tribromophenol	79 %	10 to 123
##Terphenyl-d14	74 %	33 to 141

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

J Indicates detected but below method detection limits

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-B5C
ETL Sample # : 94120941
Concentration Units: ug/L
Method: 8270TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/04/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
1,4-Dichlorobenzene	ND	40
Total Cresol	ND	40
Hexachloroethane	ND	40
Nitrobenzene	ND	40
Hexachlorobutadiene	ND	40
2,4,6-Trichlorophenol	ND	40
2,4,5-Trichlorophenol	ND	40
2,4-Dinitrotoluene	ND	40
Hexachlorobenzene	ND	40
Pentachlorophenol	ND	200
Pyridine	ND	80
##2-Fluorophenol	64 %	21 to 100
##Phenol-d6	73 %	10 to 94
##Nitrobenzene-d5	79 %	35 to 114
##2-Fluorobiphenyl	79 %	43 to 116
##2,4,6-Tribromophenol	98 %	10 to 123
##Terphenyl-d14	86 %	33 to 141

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

J Indicates detected but below method detection limits

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-ZZ3
ETL Sample # : 94120942
Concentration Units: ug/L
Method: 8270TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/04/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
1,4-Dichlorobenzene	ND	40
Total Cresol	ND	40
Hexachloroethane	ND	40
Nitrobenzene	ND	40
Hexachlorobutadiene	ND	40
2,4,6-Trichlorophenol	ND	40
2,4,5-Trichlorophenol	ND	40
2,4-Dinitrotoluene	ND	40
Hexachlorobenzene	ND	40
Pentachlorophenol	ND	200
Pyridine	ND	80
##2-Fluorophenol	62 %	21 to 100
##Phenol-d6	70 %	10 to 94
##Nitrobenzene-d5	72 %	35 to 114
##2-Fluorobiphenyl	70 %	43 to 116
##2,4,6-Tribromophenol	96 %	10 to 123
##Terphenyl-d14	82 %	33 to 141

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLP-D2
ETL Sample # : 94120943
Concentration Units: ug/L
Method: 8270TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/04/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
1,4-Dichlorobenzene	ND	40
Total Cresol	ND	40
Hexachloroethane	ND	40
Nitrobenzene	ND	40
Hexachlorobutadiene	ND	40
2,4,6-Trichlorophenol	ND	40
2,4,5-Trichlorophenol	ND	40
2,4-Dinitrotoluene	ND	40
Hexachlorobenzene	ND	40
Pentachlorophenol	ND	200
Pyridine	ND	80
##2-Fluorophenol	68 %	21 to 100
##Phenol-d6	75 %	10 to 94
##Nitrobenzene-d5	77 %	35 to 114
##2-Fluorobiphenyl	75 %	43 to 116
##2,4,6-Tribromophenol	97 %	10 to 123
##Terphenyl-d14	82 %	33 to 141

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

J Indicates detected but below method detection limits

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: EXTRACT BLANK
ETL Sample # : 95010309
Concentration Units: ug/L
Method: 8270TCLP

Date Sampled : 1/04/1995
Date Received:
Date Analyzed: 1/04/1994
Sample Diln. Fx: 1.00

Compound	Result	MDL
1,4-Dichlorobenzene	ND	10
Total Cresol	ND	10
Hexachloroethane	ND	10
Nitrobenzene	ND	10
Hexachlorobutadiene	ND	10
2,4,6-Trichlorophenol	ND	10
2,4,5-Trichlorophenol	ND	10
2,4-Dinitrotoluene	ND	10
Hexachlorobenzene	ND	10
Pentachlorophenol	ND	50
Pyridine	ND	20
##2-Fluorophenol	55 %	21 to 100
##Phenol-d6	57 %	10 to 94
##Nitrobenzene-d5	60 %	35 to 114
##2-Fluorobiphenyl	59 %	43 to 116
##2,4,6-Tribromophenol	77 %	10 to 123
##Terphenyl-d14	64 %	33 to 141

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
J Indicates detected but below method detection limits
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLPMS SA# 94120943
ETL Sample # : 95010024
Concentration Units: %
Method: 8270X

Date Sampled : 1/03/1995
Date Received: 1/03/1995
Date Analyzed: 1/04/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
Semivolatiles - TCLPMS	92	N/A

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

SEMIVOLATILE ORGANICS ANALYSIS DATA SHEET

Sample ID: TCLPMS SA# 94120943
ETL Sample # : 95010024
Concentration Units: %
Method: 8270

Date Sampled : 1/03/1995
Date Received: 1/03/1995
Date Analyzed: 1/04/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
##2-Fluorophenol	63 %	21 to 100
##Phenol-d6	65 %	10 to 94
##Nitrobenzene-d5	78 %	35 to 114
##2-Fluorobiphenyl	70 %	43 to 116
##2,4,6-Tribromophenol	94 %	10 to 123
##Terphenyl-d14	84 %	33 to 141

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

J Indicates detected but below method detection limits

B Indicates analyte detected in blank at a concentration equal to or greater than 10% of the analyte concentration in the sample

MDL Method Detection Limit

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-N45
ETL Sample # : 94120934
Concentration Units: ug/L
Method: 8081TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/07/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
Gamma BHC(Lindane)	ND	0.021
Heptachlor	ND	0.029
Heptachlor epoxide	ND	0.056
Endrin	ND	0.014
Methoxychlor	ND	0.036
Chlordane	ND	1.0
Toxaphene	ND	0.36
##Tetrachloro-m-xylene	72 %	60 to 150
##Decachlorobiphenyl	83 %	60 to 150

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

B Indicates analyte detected in blank at a concentration equal to or greater than 10% of the analyte concentration in the sample

MDL Method Detection Limit

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-044
ETL Sample # : 94120935
Concentration Units: ug/L
Method: 8081TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/07/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
Gamma BHC(Lindane)	ND	0.021
Heptachlor	ND	0.029
Heptachlor epoxide	ND	0.056
Endrin	ND	0.014
Methoxychlor	ND	0.036
Chlordane	ND	1.0
Toxaphene	ND	0.36
##Tetrachloro-m-xylene	82 %	60 to 150
##Decachlorobiphenyl	88 %	60 to 150

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-P43
ETL Sample # : 94120936
Concentration Units: ug/L
Method: 8081TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/07/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
Gamma BHC(Lindane)	ND	0.021
Heptachlor	ND	0.029
Heptachlor epoxide	ND	0.056
Endrin	ND	0.014
Methoxychlor	ND	0.036
Chlordane	ND	1.0
Toxaphene	ND	0.36
##Tetrachloro-m-xylene	84 %	60 to 150
##Decachlorobiphenyl	80 %	60 to 150

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-C3
ETL Sample # : 94120937
Concentration Units: ug/L
Method: 8081TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/07/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
Gamma BHC(Lindane)	ND	0.021
Heptachlor	ND	0.029
Heptachlor epoxide	ND	0.056
Endrin	ND	0.014
Methoxychlor	ND	0.036
Chlordane	ND	1.0
Toxaphene	ND	0.36
##Tetrachloro-m-xylene	81 %	60 to 150
##Decachlorobiphenyl	85 %	60 to 150

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-D3
ETL Sample # : 94120938
Concentration Units: ug/L
Method: 8081TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/07/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
Gamma BHC(Lindane)	ND	0.021
Heptachlor	ND	0.029
Heptachlor epoxide	ND	0.056
Endrin	ND	0.014
Methoxychlor	ND	0.036
Chlordane	ND	1.0
Toxaphene	ND	0.36
##Tetrachloro-m-xylene	90 %	60 to 150
##Decachlorobiphenyl	80 %	60 to 150

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-B5G
ETL Sample # : 94120939
Concentration Units: ug/L
Method: 8081TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/07/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
Gamma BHC(Lindane)	ND	0.021
Heptachlor	ND	0.029
Heptachlor epoxide	ND	0.056
Endrin	ND	0.014
Methoxychlor	ND	0.036
Chlordane	ND	1.0
Toxaphene	ND	0.36
##Tetrachloro-m-xylene	87 %	60 to 150
##Decachlorobiphenyl	81 %	60 to 150

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-C2
ETL Sample # : 94120940
Concentration Units: ug/L
Method: 8081TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/07/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
Gamma BHC(Lindane)	ND	0.021
Heptachlor	ND	0.029
Heptachlor epoxide	ND	0.056
Endrin	ND	0.014
Methoxychlor	ND	0.036
Chlordane	ND	1.0
Toxaphene	ND	0.36
##Tetrachloro-m-xylene	61 %	60 to 150
##Decachlorobiphenyl	87 %	60 to 150

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-B5C
ETL Sample # : 94120941
Concentration Units: ug/L
Method: 8081TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/07/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
Gamma BHC(Lindane)	ND	0.021
Heptachlor	ND	0.029
Heptachlor epoxide	ND	0.056
Endrin	ND	0.014
Methoxychlor	ND	0.036
Chlordane	ND	1.0
Toxaphene	ND	0.36
##Tetrachloro-m-xylene	71 %	60 to 150
##Decachlorobiphenyl	84 %	60 to 150

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-ZZ3
ETL Sample # : 94120942
Concentration Units: ug/L
Method: 8081TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/07/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
Gamma BHC(Lindane)	ND	0.021
Heptachlor	ND	0.029
Heptachlor epoxide	ND	0.056
Endrin	ND	0.014
Methoxychlor	ND	0.036
Chlordane	ND	1.0
Toxaphene	ND	0.36
##Tetrachloro-m-xylene	76 %	60 to 150
##Decachlorobiphenyl	85 %	60 to 150

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-D2
ETL Sample # : 94120943
Concentration Units: ug/L
Method: 8081TCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/07/1995
Sample Diln. Fx: 4.00

Compound	Result	MDL
Gamma BHC(Lindane)	ND	0.021
Heptachlor	ND	0.029
Heptachlor epoxide	ND	0.056
Endrin	ND	0.014
Methoxychlor	ND	0.036
Chlordane	ND	1.0
Toxaphene	ND	0.36
##Tetrachloro-m-xylene	81 %	60 to 150
##Decachlorobiphenyl	89 %	60 to 150

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLPMS SA# 94120943
ETL Sample # : 95010024
Concentration Units: %
Method: 8081

Date Sampled : 1/03/1995
Date Received: 1/03/1995
Date Analyzed: 1/07/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
Pesticides - TCLPMS	84	N/A

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

PESTICIDES ANALYSIS DATA SHEET

Sample ID: TCLPMS SA# 94120943
ETL Sample # : 95010024
Concentration Units: %
Method: 8081

Date Sampled : 1/03/1995
Date Received: 1/03/1995
Date Analyzed: 1/07/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
##Tetrachloro-m-xylene	87 %	60 to 150
##Decachlorobiphenyl	129 %	60 to 150

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

PESTICIDES ANALYSIS DATA SHEET

Sample ID: EXTRACT BLANK
ETL Sample # : 95010366
Concentration Units: ug/L
Method: 8081TCLP

Date Sampled : 1/04/1995
Date Received:
Date Analyzed: 1/06/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
Gamma BHC(Lindane)	ND	0.0052
Heptachlor	ND	0.0073
Heptachlor epoxide	ND	0.014
Endrin	ND	0.0034
Methoxychlor	ND	0.0090
Chlordane	ND	0.26
Toxaphene	ND	0.090
##Tetrachloro-m-xylene	92 %	60 to 150
##Decachlorobiphenyl	104 %	60 to 150

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-N45
ETL Sample # : 94120934
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/11/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
2,4-D	ND	1.5
2,4,5-TP(Silvex)	ND	0.43
##2,4-Dichlorophenylacetic acid	71 %	48 to 152

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-044
ETL Sample # : 94120935
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/11/1995
Sample Diln. Fx: 20.00

Compound	Result	MDL
2,4-D	ND	3.0
2,4,5-TP(Silvex)	ND	0.86
##2,4-Dichlorophenylacetic acid	87 %	48 to 152

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-P43
ETL Sample # : 94120936
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/11/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
2,4-D	ND	1.5
2,4,5-TP(Silvex)	ND	0.43
##2,4-Dichlorophenylacetic acid	49 %	48 to 152

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-C3
ETL Sample # : 94120937
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/11/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
2,4-D	ND	1.5
2,4,5-TP(Silvex)	ND	0.43
##2,4-Dichlorophenylacetic acid	51 %	48 to 152

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-D3
ETL Sample # : 94120938
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/11/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
2,4-D	ND	1.5
2,4,5-TP(Silvex)	ND	0.43
##2,4-Dichlorophenylacetic acid	54 %	48 to 152

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-B5G
ETL Sample # : 94120939
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/12/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
2,4-D	ND	1.5
2,4,5-TP(Silvex)	ND	0.43
##2,4-Dichlorophenylacetic acid	56 %	48 to 152

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-C2
ETL Sample # : 94120940
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/12/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
2,4-D	ND	1.5
2,4,5-TP(Silvex)	ND	0.43
##2,4-Dichlorophenylacetic acid	54 %	48 to 152

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-B5C
ETL Sample # : 94120941
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/12/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
2,4-D	ND	1.5
2,4,5-TP(Silvex)	ND	0.43
##2,4-Dichlorophenylacetic acid	54 %	48 to 152

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-ZZ3
ETL Sample # : 94120942
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/12/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
2,4-D	ND	1.5
2,4,5-TP(Silvex)	ND	0.43
##2,4-Dichlorophenylacetic acid	63 %	48 to 152

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLP-D2
ETL Sample # : 94120943
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 12/27/1994
Date Received: 12/28/1994
Date Analyzed: 1/12/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
2,4-D	ND	1.5
2,4,5-TP(Silvex)	ND	0.43
##2,4-Dichlorophenylacetic acid	56 %	48 to 152

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLPMS SA# 94120943
ETL Sample # : 95010024
Concentration Units: %
Method: 6640B

Date Sampled : 1/03/1995
Date Received: 1/03/1995
Date Analyzed: 1/12/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
Herbicides TCLPMS	74	N/A

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: TCLPMS SA# 94120943
ETL Sample # : 95010024
Concentration Units: %
Method: 6640B

Date Sampled : 1/03/1995
Date Received: 1/03/1995
Date Analyzed: 1/12/1995
Sample Diln. Fx: 1.00

Compound	Result	MDL
##2,4-Dichlorophenylacetic acid	108 %	48 to 152

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: EXTRACT BLANK
ETL Sample # : 95010369
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 1/06/1995
Date Received:
Date Analyzed: 1/11/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
2,4-D	ND	1.5
2,4,5-TP(Silvex)	ND	0.43
##2,4-Dichlorophenylacetic acid	33 %*	48 to 152

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

HERBICIDES ANALYSIS DATA SHEET

Sample ID: EXTRACT BLANK
ETL Sample # : 95010371
Concentration Units: ug/L
Method: 6640BTCLP

Date Sampled : 1/09/1995
Date Received:
Date Analyzed: 1/12/1995
Sample Diln. Fx: 10.00

Compound	Result	MDL
2,4-D	ND	1.5
2,4,5-TP(Silvex)	ND	0.43
##2,4-Dichlorophenylacetic acid	60 %	48 to 152

Surrogate
* Outside QC limits
ND Not Detected
N/A Not Applicable
B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

INORGANIC ANALYSIS DATA SHEET

Sample ID: TCLP-N45

Date Sampled : 12/27/1994

ETL Sample # : 94120934

Date Received: 12/28/1994

Compound	Method	Result	MDL	Units	Date Analyzed
% Solids	3540B,7.2	90	N/A	w/w	1/02/1995
Total Arsenic	6010TCLP	ND	220	ug/L	1/11/1995
Total Barium	6010TCLP	1300	41	ug/L	1/11/1995
Total Cadmium	6010TCLP	56	28	ug/L	1/11/1995
Total Chromium	6010TCLP	ND	98	ug/L	1/11/1995
Total Lead	6010TCLP	ND	280	ug/L	1/11/1995
Total Mercury	7471TCLP	ND	10	ug/L	1/10/1995
Total Selenium	6010TCLP	ND	620	ug/L	1/11/1995
Total Silver	6010TCLP	ND	54	ug/L	1/11/1995

ND Not Detected at or above method detection limit
N/A Not Applicable
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

INORGANIC ANALYSIS DATA SHEET

Sample ID: TCLP-O44

Date Sampled : 12/27/1994

ETL Sample # : 94120935

Date Received: 12/28/1994

Compound	Method	Result	MDL	Units	Date Analyzed
% Solids	3540B,7.2	82	N/A	w/w	1/02/1995
Total Arsenic	6010TCLP	ND	220	ug/L	1/11/1995
Total Barium	6010TCLP	1300	41	ug/L	1/11/1995
Total Cadmium	6010TCLP	47	28	ug/L	1/11/1995
Total Chromium	6010TCLP	ND	98	ug/L	1/11/1995
Total Lead	6010TCLP	ND	280	ug/L	1/11/1995
Total Mercury	7471TCLP	ND	10	ug/L	1/10/1995
Total Selenium	6010TCLP	ND	620	ug/L	1/11/1995
Total Silver	6010TCLP	ND	54	ug/L	1/11/1995

ND Not Detected at or above method detection limit
N/A Not Applicable
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

INORGANIC ANALYSIS DATA SHEET

Sample ID: TCLP-P43

Date Sampled : 12/27/1994

ETL Sample # : 94120936

Date Received: 12/28/1994

Compound	Method	Result	MDL	Units	Date Analyzed
% Solids	3540B,7.2	90	N/A	w/w	1/02/1995
Total Arsenic	6010TCLP	ND	220	ug/L	1/11/1995
Total Barium	6010TCLP	1000	41	ug/L	1/11/1995
Total Cadmium	6010TCLP	47	28	ug/L	1/11/1995
Total Chromium	6010TCLP	140	98	ug/L	1/11/1995
Total Lead	6010TCLP	ND	280	ug/L	1/11/1995
Total Mercury	7471TCLP	ND	10	ug/L	1/10/1995
Total Selenium	6010TCLP	ND	620	ug/L	1/11/1995
Total Silver	6010TCLP	ND	54	ug/L	1/11/1995

ND Not Detected at or above method detection limit
N/A Not Applicable
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

INORGANIC ANALYSIS DATA SHEET

Sample ID: TCLP-C3

Date Sampled : 12/27/1994

ETL Sample # : 94120937

Date Received: 12/28/1994

Compound	Method	Result	MDL	Units	Date Analyzed
% Solids	3540B,7.2	88	N/A	w/w	1/02/1995
Total Arsenic	6010TCLP	ND	220	ug/L	1/11/1995
Total Barium	6010TCLP	1200	41	ug/L	1/11/1995
Total Cadmium	6010TCLP	43	28	ug/L	1/11/1995
Total Chromium	6010TCLP	ND	98	ug/L	1/11/1995
Total Lead	6010TCLP	ND	280	ug/L	1/11/1995
Total Mercury	7471TCLP	ND	10	ug/L	1/10/1995
Total Selenium	6010TCLP	ND	620	ug/L	1/11/1995
Total Silver	6010TCLP	ND	54	ug/L	1/11/1995

ND Not Detected at or above method detection limit
N/A Not Applicable
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

INORGANIC ANALYSIS DATA SHEET

Sample ID: TCLP-D3

Date Sampled : 12/27/1994

ETL Sample # : 94120938

Date Received: 12/28/1994

Compound	Method	Result	MDL	Units	Date Analyzed
% Solids	3540B,7.2	86	N/A	w/w	1/02/1995
Total Arsenic	6010TCLP	ND	220	ug/L	1/11/1995
Total Barium	6010TCLP	1000	41	ug/L	1/11/1995
Total Cadmium	6010TCLP	43	28	ug/L	1/11/1995
Total Chromium	6010TCLP	ND	98	ug/L	1/11/1995
Total Lead	6010TCLP	730	280	ug/L	1/11/1995
Total Mercury	7471TCLP	ND	10	ug/L	1/10/1995
Total Selenium	6010TCLP	ND	620	ug/L	1/11/1995
Total Silver	6010TCLP	ND	54	ug/L	1/11/1995

ND Not Detected at or above method detection limit
N/A Not Applicable
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

INORGANIC ANALYSIS DATA SHEET

Sample ID: TCLP-B5G

Date Sampled : 12/27/1994

ETL Sample # : 94120939

Date Received: 12/28/1994

Compound	Method	Result	MDL	Units	Date Analyzed
% Solids	3540B,7.2	90	N/A	w/w	1/02/1995
Total Arsenic	6010TCLP	ND	220	ug/L	1/11/1995
Total Barium	6010TCLP	2600	41	ug/L	1/11/1995
Total Cadmium	6010TCLP	40	28	ug/L	1/11/1995
Total Chromium	6010TCLP	ND	98	ug/L	1/11/1995
Total Lead	6010TCLP	ND	280	ug/L	1/11/1995
Total Mercury	7471TCLP	ND	10	ug/L	1/10/1995
Total Selenium	6010TCLP	ND	620	ug/L	1/11/1995
Total Silver	6010TCLP	ND	54	ug/L	1/11/1995

ND Not Detected at or above method detection limit
N/A Not Applicable
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

INORGANIC ANALYSIS DATA SHEET

Sample ID: TCLP-C2

Date Sampled : 12/27/1994

ETL Sample # : 94120940

Date Received: 12/28/1994

Compound	Method	Result	MDL	Units	Date Analyzed
% Solids	3540B,7.2	83	N/A	w/w	1/02/1995
Total Arsenic	6010TCLP	ND	220	ug/L	1/11/1995
Total Barium	6010TCLP	1400	41	ug/L	1/11/1995
Total Cadmium	6010TCLP	90	28	ug/L	1/11/1995
Total Chromium	6010TCLP	100	98	ug/L	1/11/1995
Total Lead	6010TCLP	830	280	ug/L	1/11/1995
Total Mercury	7471TCLP	ND	10	ug/L	1/10/1995
Total Selenium	6010TCLP	ND	620	ug/L	1/11/1995
Total Silver	6010TCLP	ND	54	ug/L	1/11/1995

ND Not Detected at or above method detection limit
N/A Not Applicable
MDL Method Detection Limit

INORGANIC ANALYSIS DATA SHEET

Sample ID: TCLP-B5C

Date Sampled : 12/27/1994

ETL Sample # : 94120941

Date Received: 12/28/1994

Compound	Method	Result	MDL	Units	Date Analyzed
% Solids	3540B,7.2	86	N/A	w/w	1/02/1995
Total Arsenic	6010TCLP	ND	220	ug/L	1/11/1995
Total Barium	6010TCLP	930	41	ug/L	1/11/1995
Total Cadmium	6010TCLP	48	28	ug/L	1/11/1995
Total Chromium	6010TCLP	ND	98	ug/L	1/11/1995
Total Lead	6010TCLP	340	280	ug/L	1/11/1995
Total Mercury	7471TCLP	ND	10	ug/L	1/10/1995
Total Selenium	6010TCLP	ND	620	ug/L	1/11/1995
Total Silver	6010TCLP	ND	54	ug/L	1/11/1995

ND Not Detected at or above method detection limit
N/A Not Applicable
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

INORGANIC ANALYSIS DATA SHEET

Sample ID: TCLP-ZZ3

Date Sampled : 12/27/1994

ETL Sample # : 94120942

Date Received: 12/28/1994

Compound	Method	Result	MDL	Units	Date Analyzed
% Solids	3540B,7.2	78	N/A	w/w	1/02/1995
Total Arsenic	6010TCLP	ND	220	ug/L	1/11/1995
Total Barium	6010TCLP	590	41	ug/L	1/11/1995
Total Cadmium	6010TCLP	42	28	ug/L	1/11/1995
Total Chromium	6010TCLP	130	98	ug/L	1/11/1995
Total Lead	6010TCLP	4300	280	ug/L	1/11/1995
Total Mercury	7471TCLP	ND	10	ug/L	1/10/1995
Total Selenium	6010TCLP	ND	620	ug/L	1/11/1995
Total Silver	6010TCLP	ND	54	ug/L	1/11/1995

ND Not Detected at or above method detection limit
N/A Not Applicable
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

INORGANIC ANALYSIS DATA SHEET

Sample ID: TCLP-D2

Date Sampled : 12/27/1994

ETL Sample # : 94120943

Date Received: 12/28/1994

Compound	Method	Result	MDL	Units	Date Analyzed
% Solids	3540B,7.2	84	N/A	w/w	1/02/1995
Total Arsenic	6010TCLP	ND	220	ug/L	1/11/1995
Total Barium	6010TCLP	1300	41	ug/L	1/11/1995
Total Cadmium	6010TCLP	47	28	ug/L	1/11/1995
Total Chromium	6010TCLP	ND	98	ug/L	1/11/1995
Total Lead	6010TCLP	300	280	ug/L	1/11/1995
Total Mercury	7471TCLP	ND	10	ug/L	1/10/1995
Total Selenium	6010TCLP	ND	620	ug/L	1/11/1995
Total Silver	6010TCLP	ND	54	ug/L	1/11/1995

ND Not Detected at or above method detection limit
N/A Not Applicable
MDL Method Detection Limit

REPORT NUMBER: 94J-1005

Ciba - Geigy Corporation
ENVIRONMENTAL TESTING LABORATORY

INORGANIC ANALYSIS DATA SHEET

Sample ID: TCLPMS SA# 94120943

Date Sampled : 1/03/1995

ETL Sample # : 95010024

Date Received: 1/03/1995

Compound	Method	Result	MDL	Date Analyzed
Total Mercury - TCLPMS	7471	70 %	N/A	1/10/1995
Total Silver - TCLPMS	200.7	89 %	N/A	1/11/1995
Total Arsenic - TCLPMS	200.7	93 %	N/A	1/11/1995
Total Barium - TCLPMS	200.7	94 %	N/A	1/11/1995
Total Cadmium - TCLPMS	200.7	90 %	N/A	1/11/1995
Total Chromium - TCLPMS	200.7	90 %	N/A	1/11/1995
Total Lead - TCLPMS	200.7	89 %	N/A	1/11/1995
Total Selenium - TCLPMS	200.7	100 %	N/A	1/11/1995

Surrogate

* Outside QC limits

ND Not Detected

N/A Not Applicable

B Indicates analyte detected in blank at a concentration
equal to or greater than 10% of the analyte concentration
in the sample

MDL Method Detection Limit

SL SAVANNAH LABORATORIES & ENVIRONMENTAL SERVICES, INC.

5102 LaRoche Avenue • Savannah, GA 31404 • (912) 354-7858 • Fax (912) 352-0165

LOG NO: S4-47012

Received: 29 DEC 94

Ms. Julie Smith
Ciba Geigy Environmental Testing
P.O. Box 71
Toms River, NJ 08754

Purchase Order: L 90179

Project: CRN-TCLP (S)
Sampled By: Client

REPORT OF RESULTS

Page 1

LOG NO	SAMPLE DESCRIPTION , SOLID OR SEMISOLID SAMPLES				DATE SAMPLED
47012-1	TCLP-N45				12-27-94
47012-2	TCLP-044				12-27-94
47012-3	TCLP-P43				12-27-94
47012-4	TCLP-C3				12-27-94
47012-5	TCLP-D3				12-27-94
PARAMETER	47012-1	47012-2	47012-3	47012-4	47012-5
Total Releasable Cyanide, mgHCN/kg	<1.0	<1.0	<1.0	<1.0	<1.0
Total Releasable Sulfide, mgH ₂ S/kg waste	<10	<10	<10	<10	<10
Corrosivity-pH (EPA 9045), units	8.04	7.94	7.91	7.86	7.58
Ignitability-flash point, Degrees F	*F6	*F6	*F6	*F6	*F6
Percent Solids, %	87	83	88	84	87

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REPORT OF RESULTS

Page 2

LOG NO	SAMPLE DESCRIPTION , SOLID OR SEMISOLID SAMPLES				DATE SAMPLED
47012-6	TCLP-B5G				12-27-94
47012-7	TCLP-C2				12-27-94
47012-8	TCLP-B5C				12-27-94
47012-9	TCLP-ZZ3				12-27-94
47012-10	TCLP-D2				12-27-94
PARAMETER	47012-6	47012-7	47012-8	47012-9	47012-10
Total Releasable Cyanide, mgHCN/kg	<1.0	<1.0	<1.0	<1.0	<1.0
Total Releasable Sulfide, mgH ₂ S/kg waste	<10	<10	<10	<10	<10
Corrosivity-pH (EPA 9045), units	7.08	7.18	7.26	6.38	6.30
Ignitability-flash point, Degrees F	*F6	*F6	*F6	*F6	*F6
Percent Solids, %	89	81	86	85	83

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REPORT OF RESULTS

Page 3

LOG NO SAMPLE DESCRIPTION , QC REPORT FOR SOLID/SEMISOLID

47012-11 Method Blank
47012-12 LCS/LCS Duplicate % Recovery
47012-13 LCS % RPD
47012-14 Date Analyzed

PARAMETER	47012-11	47012-12	47012-13	47012-14
Total Releasable Cyanide, mgHCN/kg	<1.0	---	0 %	01.02.95
Total Releasable Sulfide, mgH ₂ S/kg waste	<10	---	0 %	12.30.94
Corrosivity-pH (EPA 9045), units	---	98/98 %	0 %	01.06.95
Ignitability-flash point	---	---	---	01.11/12

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Purchase Order: L 90179

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Sampled By: Client

REPORT OF RESULTS

Page 4

LOG NO SAMPLE DESCRIPTION , QC REPORT FOR SOLID/SEMISOLID

47012-15 Date Report Submitted

PARAMETER 47012-15

Date Reported 01.12.95

Methods: EPA SW-846

*F6 = The physical characteristics (hard, semisolid) of the sample prohibited continual stirring required by the Pensky-Martens Method. Therefore, the sample was heated to >140 F without continual stirring and a test flame applied to the sample surface. No apparent ignition of vapors over the sample was observed.

Linda A. Wolfe
Linda A. Wolfe

Final Page Of Report

SL SAVANNAH LABORATORIES & ENVIRONMENTAL SERVICES, INC.

5102 LaRoche Avenue • Savannah, GA 31404 • (912) 354-7858 • Fax (912) 352-0165

REMIT TO: P.O. Box 13548, Savannah, Georgia 31416-0548

CLIENT PO. NO: L 90179

PROJECT NO: CRN-TCLP (S)

Ms. Julie Smith
Ciba Geigy Environmental Testing
P.O. Box 71
Toms River, NJ 08754

INVOICE NO: S50155

INVOICE DATE: 12 JAN 1995

INVOICE CC: Diana Baldi

TERMS: Net 30 Days

Federal Tax ID NO: 58-1485724

LOG NO: S447012

INVOICE

CODE: CIBA-NJ-S-60LW

ITEM	SAMPLE IDENTIFICATION	QTY	ANALYSIS	PRICE	AMOUNT
1	TCLP-N45 TCLP-044 TCLP-P43 TCLP-C3 TCLP-D3 TCLP-B5G TCLP-C2 TCLP-B5C TCLP-ZZ3 TCLP-D2	10	Total Releasable Cyanide Total Releasable Sulfide Corrosivity-pH (EPA 9045) Ignitability-flash point Percent Solids	\$142.50	1,425.00
2	Method Blank LCS/LCS Duplicate & Recovery LCS & RPD Date Analyzed	4	Total Releasable Cyanide Total Releasable Sulfide Corrosivity-pH (EPA 9045) Ignitability-flash point		
	Date Report Submitted	1	Date Reported		

TOTAL

\$1,425.00

REPORTED TO: Ms. Julie Smith

CUSTOMER PHONE: 908/914-2845

For Proper Credit, please show INVOICE NUMBER on your remittance.

After 30 days, service charges of 1.5% per 30 days will be applied to unpaid balance.

CERTIFIED LAB # 297

AL CERTIFIED LAB # 40590

CT CERTIFIED LAB # PH 0705

IA CERTIFIED # 101

SC CERTIFIED LAB # 94011

PA CERTIFIED LAB # 68-452

MA CERTIFIED LAB # NJ254

DE CERTIFIED LAB

CORPORATE LABORATORY

CHAIN OF CUSTODY

ROUTE 37 W TOMS RIVER, NJ 08754

(908) 914-2500 or (800) 962-6933

FAX (908) 914-2916



PAGE 1 OF 1

SHADED AREA 0842500326 FOR ETL USE ONLY			INFORMATION PROVIDED BY CLIENT														
			SAMPLING INFORMATION					CONTAINER TYPE, SIZE, QUANTITY AND PRESERVATIVE									
PROJECT CODE: CRP-TCLP(S)	PROJECT REQUEST: 4137		SAMPLED BY: KENNETH A. KENT/SIGNATURE: <i>[Signature]</i>					500ml W/m	250ml W/M								
CHARGE NUMBER: CLA-61	JOB NUMBER: 94J- 1005		TITLE: SENIOR TECHNICIAN COMPANY: Ciba, Cranston (WCC)					Clear Glass	Clear Glass								
T-A-T: 1-16-95 10D PR 1/2/95			PHONE NUMBER (201) 785-0700 EXT. 295					Jar un- pres erved	Jar un- pres erved								
LAB NUMBER	DATE	TIME	MATRIX	SAMPLE DESCRIPTION		TEST/COMMENTS											
94120934	12/27/94	1000	Solid	TCLP - N45		SP-TCLP, * RCRA		1	1								
0935	"	1020	"	TCLP - 044		"		1	1								
0936	"	1030	"	TCLP - P43		"		1	1								
0937	"	1100	"	TCLP - C3		"		1	1								
0938	"	1115	"	TCLP - D3		"		1	1								
0939	"	1130	"	TCLP - B5G		"		1	1								
0940	"	1145	"	TCLP - C2		"		1	1								
0941	"	1220	"	TCLP - B5C		"		1	1								
0942	"	1240	"	TCLP - Z23		"		1	1								
0943	"	1300	"	TCLP - D2		"		1	1								
CUSTODY SEAL INTACT? <input checked="" type="checkbox"/> Y <input type="checkbox"/> N/A			RELINQUISHED BY: (Signature) <i>[Signature]</i>		DATE	TIME	RECEIVED FOR LABORATORY BY: (Signature) <i>[Signature]</i>		DATE:		TIME:						
SAMPLES PRESERVED? <input checked="" type="checkbox"/> Y <input type="checkbox"/> N/A			RECEIVED BY: (Signature) <i>[Signature]</i>		12/27/94	1400	REMARKS: <i>[Signature]</i>		12-28-94		1000						
HEADSPACE IN VOA'S? <input checked="" type="checkbox"/> Y <input type="checkbox"/> N/A																	
VOA TEFALON BIDE DOWN? <input checked="" type="checkbox"/> Y <input type="checkbox"/> N/A																	
COOLER TEMPERATURE: 3°C			RELINQUISHED BY: (Signature)														
COMMENTS:			RECEIVED BY: (Signature)														